BUREAU OF HYGIENE AND TROPICAL DISEASES.

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## AMOEBIASIS AND DYSENTERY.

### Amoebiasis.

James (W. M.). **A Survey for Amoebiasis at Santa Marta, Colombia. (Preliminary Report.)**—*Fourteenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1925. pp. 160–165.

The author lays stress on the necessity in all doubtful cases of examining a permanently mounted wet-fixed and stained preparation ; in such a preparation identification of *E. histolytica* is certain, whereas it is often doubtful in fresh and iodine preparations. Permanent preparations will also invariably show a higher incidence of all kinds of entamoebae than will fresh or iodine preparations. From one case four fresh stools were absolutely negative even in the fixed and stained preparations ; yet the fifth stool showed numerous *E. histolytica*. In suspected cases, therefore, re-examination of stools is obligatory. The examination of stool specimens for *E. histolytica* cannot be considered as a light routine laboratory procedure, if correct diagnosis is desired. The author is sure that in the old days at Ancon Hospital an enormous number of amoebic infections were overlooked. There must be a thoroughly trained personnel to conduct stool examinations.

*Results of survey.*—Over 50 per cent. of hospital patients proved to be infected with *E. histolytica*, and the hospital personnel revealed almost as high an incidence. The Fruit Company staff (other than hospital) gave an incidence of about 10 per cent. less than the above two groups. The club servants, waiters, cooks, revealed the astonishing incidence of over 60 per cent. The outpatient group, all with histories of past infection, were more than 60 per cent. infected. Amoebic is far more widespread than any other form of protozoal infection in the Tropics. Even if a *winter season* should come to the Tropics the incidence of amoebic infection would remain unchanged so long as the population remained filthy in their habits. The damage done by a long-continued chronic amoebic infection is worse than that inflicted by chronic malaria ; chronic amoebic infection is nearly always associated with intestinal worms.

*Method of infection.*—The author is emphatic that this is always—" *a method of finger to mouth, directly or indirectly.*"

*Prevention.*—" *The best preventive measure is to insist that all servants clean their hands thoroughly with scrubbing brush, antiseptic soap, and water several times a day—especially before preparing or serving food !* "

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With Dr. KOFOID, the author has noted how entamoebae disappear from the stools of cases subjected to vigorous bismuth treatment. He believes bismuth to be the best remedy we have, but it should be given for at least six months. One must try to *eradicate the infection in the lumen of the bowel*: the tissues will have taken care of the entamoebae that have penetrated them. Dr. James declares himself still *unconvinced that amoebiasis is a genuine systemic infection.* Any lesion in a tissue that is to be considered of amoebic origin must show amoebae as entire parasites beyond any possibility of a doubt; the diagnosis cannot be established on the basis of a nuclear structure alone.

Dr. James does not accept all the species named by Dr. KOFOID, He agrees as to the morphology, but not the nomenclature, of *E. histolytica.* The entamoebae described by KOFOID as *Karyamoebina falcata* and *Councilmania lafleuri* are to James just forms of *E. coli.* In several cases where KOFOID reported *Dientamoeba fragilis,* James could find only *Endolimax nana* and *Iodamoeba bütschlii*; an opinion, he states, confirmed by Clifford DOBELL, who is quoted also as being now convinced that *E. histolytica* is *not* always a tissue parasite; it can occur in the large intestine without tissue penetration. A latent infection therefore does not necessarily mean ulceration of the bowel—though it may at any time establish an ulcerative process.

H. M. Hanschell.

KOFOID (Charles A.) & SWEZY (Olive); BOYERS (Luther M.). **A Report on an Investigation of Intestinal Protozoan Infections at Santa Marta, Colombia, in the Hospital of the United Fruit Company, with Special Reference to the Incidence of Amoebiasis.**—*Fourteenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1925. pp. 140–153. [1 ref.]

A total of 556 examinations was made on 367 persons, representing fully the native population and coastal immigrants from tropical America; these two groups consist of mixed racial stocks, Amerindian, African and Caucasian and the imported American personnel. The statistical results of these examinations are tabulated.

*E. dysenteriae* was found in 53·7 per cent.; in the staff of the United Fruit Company, 40 per cent. *Iodamoeba bütschlii* was found in 20·9 per cent. and 24 per cent. harboured *Councilmania lafleuri.* Five cases were infected with *Enteromonas hominis,* three with *Isospora hominis* and three with *Balantidium coli.* The distinct impression was conveyed by the cumulative effect of examinations that the authors were dealing with massive heavy infections in the general population. 52 per cent. had acquired 3 to 7 different parasitic infections—showing the tendency for persons exposed to faecal infections to accumulate the commoner infestations.

The epidemiology of amoebiasis is rather imperfectly known. The infected stool is the only vehicle for conveyance of contagion. To acquire amoebiasis, one must swallow something contaminated by infected faeces. WENYON and O'CONNOR showed that the housefly may retain cysts after feeding on infected faeces from five minutes to 42 hours. Fly destruction is therefore an important preventive measure, as is the careful cleansing of all foodstuffs, especially such as are eaten uncooked. The principal source of contagion, however, lies in the contacts which the infected person makes with food, utensils,

wash bowls and other necessities and conveniences which the soiled hand may contaminate.

From the authors' nine years' 52,000 examinations of over 15,000 persons, including many family groups, they find conclusive evidence that the infection tends to spread in families, it may be over three generations.

There is as yet no conclusive evidence as to the relation of amoebic infections to water supply. That the infection may be water-borne is suggested by the higher incidence of hospital cases of amoebiasis during the wet season at Santa Marta. Whether cysts survive drying and may be carried in the dust is still an open question.

In the last analysis, however, it is the *cyst distributor* who is the source of the contagion. Meanwhile " the normal history of a chronic infection of amoebiasis is one of progressive deterioration."

H. M. H.

KOFOID (Charles A.). **Human Amoebiasis with Special Reference to the Chronic Phase.**—*Texas State Jl. Med.* 1925. Oct. Vol. 21. 12 pp. [1 ref.]

In this address to the State Medical Association of Texas the author states that historically amoebiasis in man has come to be stamped with the characteristics of tropical incidence and dysenteric symptoms. This, however, is but a partial picture of this parasitic infection. In temperate regions and in the practice of most U.S. physicians this disease is only incidentally met with in its dysenteric aspect, or traced to a tropical origin. Usually it is in the chronic phase in patients who have never had tropical exposure and have no recollection of a dysentery attack. Often stasis or chronic constipation, and only rarely a diarrhoeic condition of stool, characterizes the infection. Understanding of all types of nuclear structure and all the successive changes through which the nucleus goes during nuclear division is essential for accurate diagnosis. It is not possible to establish even a probable negative by a single examination of stool. A series of six *consecutive* stools should be examined. In examining sputum for amoebic abscess of lung it is essential to distinguish motile stages of *E. dysenteriae* from motile stage of *E. gingivalis* which abounds in many mouths, especially if pyorrhoea alveolaris be present. Amoebae may also be detected in motile and encysted stages in duodenal contents obtained by drainage. Seven or more species of amoebae live in the human bowel, of which five at least may present clear pseudopodia, and at least two, *E. dysenteriae* and *Councilmania lafleuri*, may be found with red blood cells in their cytoplasm. In detection of chronic human amoebiasis, in the laboratory, he has come to rely wholly upon identification of the encysted stage. Detection of motile stages of *E. dysenteriae* in tissues rests upon the nuclear structure and appreciation of the cytological differences between amoebae and human cells.

The tissue invasive capacity of *E. dysenteriae* is demonstrated by finding the parasite, in advance of the ulceration, in the submucosa of colon, lymph glands adjacent to ulcers, and especially in the capillaries and larger blood vessels of the peripheral serosa of the colon. The amoebae are found in the portal veins, from which they invade the liver. They are found in the pus of liver abscess and in liver areas beyond the wall of the abscess. Amoebae found by duodenal drainage denote liver infection in the absence of abscess. Their entrance into general

systemic circulation is indicated by occurrence of amoebic abscess in lung and brain ; by their detection in epididymis, semen, bladder, or in the tubes. They have been reported in the skin of the perineum and in phagedenic ulcers around incision in cases of liver abscess. He has found the motile amoebae in the excised head of the femur in a case of arthritis deformans, and in the lymph glands in three cases of Hodgkin's disease.

He concludes from 46,000 examinations of 12,000 persons that these cases represent a group of persons in physicians' care with a predominance of the neurasthenic, rheumatic or intestinal type of mild invalidism, and an unusual number in whom diagnosis is obscure or complicated. In this group the incidence of infection by *E. dysenteriae* is from 10 to 20 per cent. In an examination of several groups of 60 to 100 persons not thus selected and presumably not diseased, the percentage of infected did not exceed 1 per cent. A tabulation of 7,000 persons examined indicated that those who have travelled extensively have double the rate of infection of those who have not travelled. This is indicative only of the increased risk of infection which all run who eat from many hands ; the tropics, too, are areas of lower standards of sanitation and higher incidence of infection. Amoebiasis is not limited to the tropics, but occurs widely in the human race. The nearest relatives of some of the amoebae of man are found in apes and monkeys—the human infections are the product of the evolutionary process—the parasites of man have descended from the parasites of man's ancestors. The relationships of amoebic infection, not only to dysentery, but to chronic intestinal trouble, to local abscesses, and to certain arthritic conditions, makes this infection an important one in medical practice. A thorough-going, accurate stool examination by a competent examiner trained in protozoology is highly desirable in any complete physical examination.

H. M. H.

MENK (Walther). **Amoebic Dysentery.**—*Fourteenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1925. pp. 166–169.

The author agrees with JAMES and KOFOID as to the prophylactic measures advised by them for the control of amoebic infection at Santa Marta ; if it were possible to carry them out, these measures would also reduce the incidence of enteric fevers.

He discusses generally the cases of dysentery admitted to hospital. All the patients are infected with hookworm. They cannot be kept long enough in hospital to undergo the prolonged classical treatment for amoebic dysentery, and only very few will continue any follow-up treatment outside. However, the few liver abscesses and deaths from amoebic dysentery indicate that clinical amoebic dysentery is efficiently controlled by the hospital and dispensaries medical service. A Wassermann test is indicated in all amoebic dysenteric cases that fail to respond promptly to emetine and bismuth. If the test be positive, combined antisyphilitic (preferably salvarsan) with antidysenteric treatment will generally effect a cure. Treatment for hookworm and other intestinal parasites, when such are present, should be given during a course of treatment for amoebic dysentery.

Bacillary dysentery with *E. histolytica* in the stools or acute bacillary infection engrafted on a chronic amoebic dysentery should always be

considered, especially when the symptoms are very acute with frequent foul-smelling stools, fever, vomiting, abdominal pain, dehydration and toxaemia.

No lung infection due to *E. histolytica* was observed. A series of arthritis cases, of indefinite aetiology, was treated with emetine on the chance of the cause being amoebic metastasis, involving the joints, according to the ideas of KOFOID. No marked beneficial effect from the treatment could be observed. It appears that emetine-responding arthritis is a somewhat rare condition in Santa Marta.

H. M. H.

BOYERS (Luther M.). **A Report on an Investigation of Intestinal Protozoan Infections at Santa Marta, Colombia, in the Hospital of the United Fruit Company, with Special Reference to the Incidence of Amoebiasis.**—*Fourteenth Ann. Rep. Med. Dept. United Fruit Company, Boston, Mass.* 1925. pp. 154–159. [1 ref.]

*Sigmoidoscopy.*—Most of the patients were found to have multiple infections of the bowel, in contrast with California cases where the infection is often single. The same kind of picture, however, is seen in both Colombia and California.

The picture varies according to the severity of the infection. From the erythema of mucosa in mild cases, to the severer grades of inflammation displaying dilated blood vessels with perivascular inflammation and infiltration causing the vessels to stand out boldly from surface of the mucosa; haemorrhagic areas of varying size; free blood and mucus in the lumen of the bowel; ulcerations of various size. The old chronic cases commonly showed irregular patches smoother and flatter than normal, representing apparently "scarified" areas. In a few cases, and only in those harbouring hookworm, punctate haemorrhagic areas were seen in the rectum. Pain was common in all cases—most severe in those with spastic bowel. Spasticity of rectum and sigmoid was as readily detected by sigmoidoscope as by X-ray. Haemorrhoids were common. Prolapse of rectum most often accompanied a spastic colon.

Treatment—can be divided into three periods—

(1) *Diagnostic period.*—Demonstration of presence or absence of pathogenic amoebae. Cathartics, e.g., rhubarb or magnesium sulphate, and cholagogues, e.g., *fel bovis* facilitate the search for amoebae, but in the presence of amoebiasis, since they increase hyperaemia of bowel and biliary mucosa, they are dangerous if vigorous treatment is not pressed immediately.

(2). *Eradication period.*—Attempted elimination of amoebae. The author recommends as agents to effect this all the drugs in present use, and for the case generally, digitalis, caffeine, diet, bed.

(3). *Post eradication period.*—Includes the use of a number of remedial agents, and occupational supervision, especially favouring out of doors employment.

The author has used yatren much in the past two years, and found it a useful adjunct to treatment—but has not yet obtained by it complete eradication of both motile and encysted *E. dysenteriae* in any case.

H. M. H.

CASTEX (Mariano R.). Ueber die Amöbiasis des Menschen. [**Amoebiasis in Man.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1926. Aug. 1. Vol. 30. No. 8. pp. 309–333. [46 refs.]

This abstract can give only some of the points dealt with in this interesting lecture (delivered to the medical faculty at Hamburg), covering the whole ground of the clinical manifestations and treatment of amoebiasis and the biology of the parasites. The paper is the fruit of the author's observations and researches, in collaboration with Professor Daniel GREENWAY, over a series of years, in Buenos Ayres (and already reported by them in Argentine and French medical journals). They had been the first to discover and record the occurrence of *E. histolytica* in Argentina. There it is widespread—thus in 1924, out of 614 patients with intestinal complaints 24·5 per cent. were shown to harbour *E. histolytica*; and the parasite was found in 23·6 per cent. of 2,023 such patients in 1925. These findings they attribute to the careful systematic search for the parasites; to the fact that all the patients examined were ill—simple parasite carriers were therefore completely excluded; and that these patients were adults. GREENWAY had found in one province of Argentina 1·3 per cent. of quite healthy children infested.

H. M. H.

CASTEX (M. R.) & GREENWAY (D.). [**Amebiasis in Argentina.**]—*Prensa Méd. Argentina.* Buenos Aires. 1926. Feb. 20. Vol. 12. p. 905. [Summarized in *Jl. Amer. Med. Assoc.* 1926. July 3. Vol. 87. No. 1. pp. 68–69.

An analysis of the author's experience with 479 patients with amoebiasis —forming 23·6 per cent. of the 2,023 whose stools were examined. Infestation was pure in only 92.

Patients must be made to realize that it takes months—6 to 8 in some cases—for the ulcerations to heal; and that treatment must be continued to the final extinction of the infestation, changing drugs at need. Symptomatology is protean. Each course of emetine should be followed by suspension of treatment for two or three months; emetine polyneuritis is liable to develop when least expected. In one woman amoebiasis had induced intense pruritus for years; she was unable to tolerate emetine or bismuth, and drugs per rectum caused severe colon inflammation. The infestation still persisted in her after eight months of treatment.

H. M. H.

SITSEN (A. E.). Over complicaties van de amoebiasis van den darm. [**Complications of Intestinal Amoebiasis.**]—*Geneesk. Tijdschr. v. Nederl. Indië.* 1926. Vol. 66. No. 3. pp. 343–357. [22 refs.]

Sitsen found 221 cases of intestinal amoebiasis in a series of 2,700 autopsies after death from natural causes. In 69 amoebiasis was the only apparent cause of death; in 99 other causes were present in addition to the amoebic infection; a perforation peritonitis was found in 19 cases, peritonitis without perforation in 10, abscess of the liver in 22, of the kidneys in 2 cases.

He comments especially upon the metastatic complications. It is generally accepted that the amoebae are carried by the blood; no general agreement exists, however, whether amoebic metastases beyond the liver may be caused by passage of the amoebae through the capillaries of the liver or only by perforation of a pre-existent abscess

of the liver into the hepatic venae. In many cases of pulmonary, cerebral or splenic abscess the embolic character has been shown, and the rarity of cerebral and splenic abscess is in accordance with the difficulties an embolus meets in passing the pulmonary circulation. As far as concerns amoebic bronchitis, the prevalence of which has lately been emphasized (PETZETAKIS, E. A.), Sitsen is in doubt whether the amoebae in such cases do actually belong to the histolytica type. Exogenous infection of the respiratory tract as well as of the urinary passages is possible.

Accepting that metastatic spread of amoebiasis is of embolic origin, and primarily caused by abscess of the liver, the causation of the latter is discussed. Sitsen thinks that the prevalence of the solitary abscess (in 60–75 per cent. of the cases) is in discordance with the theory of its haematogenous origin. He describes the way by which an infection may reach the liver from the caecum in a comparatively short way along the superior mesenteric glands, the subpyloric chain (against the lymph current) to the hepatic chain and the liver.

W. J. Bais.

BRUMPT (E.). Individualité de l'*Entamoeba dispar.* Présentation de pièces. [**Individuality of** *E. dispar.*]—*Bull. Soc. Path. Exot.* 1926. June 9. Vol. 19. No. 6. pp. 399–402.

WALKER and SELLARDS claim that of 20 healthy persons subjected to infestation by amoebic dysentery, four developed acute dysentery, and 14 became healthy carriers. The author's study of statistics in India, where all are exposed to natural infestation and where dysentery is widespread, show that there are 2 to 4 per cent. of dysentery cases. He reaches an identical conclusion for the French Far-Eastern colonies. Since 1914, most authors under the impression that man harbours only one amoeba producing four-nucleated cysts, and that this is the dysentery amoeba, have published statistics founded on a false basis. It is astounding that the amoebae of dysentery should cause dysentery in 20 per cent. in the Philippines, 2 to 4 per cent. in India and Indo-China, and once only in three or four million carriers in England. Liver abscess (an ailment not often unrecognized as happens in amoebic enteritis) shows a similar discordant geographical incidence. These puzzling observations would not have been published if the authors had studied the small amoeba with four-nucleated cysts described very briefly by PROWAZECK in 1912, as *E. hartmanni*; or if KUENEN and SWELLENGREBEL, and later BRUG, who had observed the same amoeba in the Dutch Indies, had been able to compare experimentally its pathogenic rôle with that of the amoeba of dysentery, or there had been confirmation of the experiments of Sohei SHIMURA, who in 1918 showed the existence in Formosa of human amoebae with four-nucleated cysts, morphologically identical with those of *E. dysenteriae,* which produced feeble and ephemeral infection in the cat.

The author's failure to infect kittens with *minuta* type amoeba from healthy carriers led him to believe that SHIMURA had been dealing with this type, which appeared to be non-pathogenic for the cat. This failure was due, however, to faulty technique, for using the discovery of SELLARDS and THEILER that cysts " hatched out " in the cat's rectum, and the technique of BOECK and DRBOHLAV (occlusion of cat's anus and production of intestinal stasis), he had obtained later a high percentage of successful infections. His experiments in 1925–26 showed that,

besides *E. dysenteriae*, there exist two other amoebae, parasitic in man, forming four-nucleated cysts, viz., *E. hartmanni*, which appears not to be inoculable into the cat, and *E. dispar*, inoculable into the cat. *E. hartmanni* forms cysts of less than 10$\mu$—easy to distinguish from those of *E. dysenteriae*. *E. dispar* forms cysts 10 to 16$\mu$, indistinguishable from those of *E. dysenteriae*. These two amoebae can be differentiated only by the fact that *E. dysenteriae* inoculated into kittens (400 to 600 grammes) produces the typical lesions of dysentery and kills them. *E. dispar*, on the contrary, though sometimes producing in the kitten a heavy infection, causes none of the lesions of dysentery. The author has studied experimentally *E. dispar* in two carriers who had travelled in various tropical lands, and in two persons who had never left France. POUBLAN has studied experimentally a fifth case in a youth of 15 years, with no intestinal trouble, who had never left France. The author is sure that C. A. HOARE (1925) obtained this same parasite in England (see below). A kitten inoculated with cysts and killed 41 days later was found to have normal intestines, no visible ulcerations, yet amoebae were demonstrated and reported as *E. dysenteriae*. If the existence of *E. dispar* be accepted, it becomes easy to interpret the surprising epidemological facts cited above.

H. M. H.

VIALARD & DARLEGUY. Sur quelques cas de pleurésie diaphragmatique droite primitive avec syndrome hépatique chez des malades coloniaux dysentériques amibiens. [**Primary Right Diaphragmatic Pleurisy in Amoebic Dysenterics from the Tropics.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1926. July 22. 3rd Ser. Year 42. Vol. 50. No. 26. pp. 1310–1318. [1 ref.]

In patients from the tropics, former sufferers from dysentery and hepatitis, who present symptoms referable to a lesion of the diaphragm, it is necessary to consider the possibility of primary pleurisy of the right base. The effect of emetine treatment will often help in the diagnosis. The differential diagnosis between suppurative hepatitis and pleural lesion of the right base may be made easier by a study of the leucocyte count. The authors' patients (primary pleurisy) all showed a mononuclear leucocytosis, as is usual in tuberculosis.

H. M. H.

FRANCHINI (Giuseppe). Sovra un caso di amebiasi generalizzata. [**A Case of Generalized Amoebiasis.**]—*Riforma Med.* 1926. Sept. 6. Vol. 42. No. 36. pp. 843–844. With 2 text figs. [5 refs.]

The author relates the case of a young woman who, three years ago, suffered from intestinal disturbance, and in whose faeces were found *E. histolytica*, both vegetative and cystic forms, and *Chilomastix mesnili*. For nearly a year the urine had been turbid, and a catheter specimen contained red corpuscles and vegetative forms of the same Entamoeba. Cure of both these conditions resulted from intramuscular injections of emetine. Three months ago she presented herself complaining of a troublesome cough, with mucopurulent expectoration, streaks of blood, and the physical signs of bronchitis. Examination of the sputum revealed vegetative forms of the Entamoeba and sparse cysts were present in the faeces. Injections of emetine were given as before, together with stovarsol by mouth. Improvement was rapid, and in a

month the patient seemed to be quite cured. The author refers to a similar case described by Mme. PANAYOTATOU (this *Bulletin,* Vol. 23, pp. 655).

H. Harold Scott.

DE VECCHI (Bindo). Amebiasi cronica.—Mesosigmoidite—Peritonite. [**Case of Chronic Amoebiasis.**]—*Pathologica.* 1926. July 15. Vol. 18. No. 417. pp. 333–335.

This is a detailed record of an interesting case of amoebiasis, calling further attention to the fact of the existence of this disease endemically in Italy.

Thirteen days before his death, a man of 65 suffered from general pains, cough, fever and dyspnoea. Abdominal symptoms, immobility and meteorism, supervened ten days later. Autopsy showed a diffuse purulent peritonitis, the splenic flexure of the colon bound down by adhesions, and in the mucosa the conditions associated with chronic amoebic infection.

H. Harold Scott.

FLANDIN (Ch.) & DUCHON (Louis). Récidive de dysenterie amibienne après trente-trois ans de guérison apparente. [**Relapse of Amoebic Dysentery 33 Years after Apparent Recovery.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1926. Aug. 5. Year 42. 3rd Ser. Vol. 50. No. 28. pp. 1416–1417.

A man, age 63 years, following fatigue and exposure to cold, developed frequent muco-sanguineous stools teeming with amoebae, together with fever, tenesmus and abdominal pain. Emetine hydrochloride 12 centigrammes a day for six days, then tréparsol 0·50 gramme a day for six days, rapidly and completely restored the patient to health and caused both amoebae and cysts to vanish from stools. A critical and close enquiry into habits, environment, friends and relatives of the patient failed to find any possible source of infection. Thirty-three years before the patient had lived in Morocco and had there suffered from dysentery, which he well remembered. The author considers that tréparsol is superior to novarsenobenzol or stovarsol in its action on amoebic cysts.

H. M. H.

SIMPSON (Alan S.). **A Case of Amoebic Dysentery with Liver Abscess contracted in England.**—*Lancet.* 1926. Sept. 4. p. 495.

The man, aged 53 years, had never been out of England. Four weeks of frequent defaecation with blood and mucus in stool, fever, 8,500 leucocytes, differential count normal. Wassermann negative, urine normal, ill defined tender tumour in right lumbar region. Stools showed vegetative and precystic forms of *E. histolytica* in large numbers. Laparotomy revealed tumour to be abscess in right hepatic lobe containing yellowish pus, sterile on culture, and showing no amoebae by microscope. Following operation, good recovery on E.B.I. gr. iii daily for 12 days.

H. M. H.

SHANKS (G.). **A Case of Amoebic Abscess of the Lung.**—*Indian Med. Gaz.* 1926. Aug. Vol. 61. No. 8. p. 393.

The case was encountered in a series of 30 autopsies on prisoners of war who died in No. 1 British General Hospital, Mesopotamia, June, 1918.

The patient was a Turkish corporal who, on admission to hospital, was emaciated and was found to be suffering from relapsing fever and amoebic dysentery. At necropsy—in *left lower lobe of lung* was an abscess 1·5 cm. diameter, near pleural surface and containing chocolate-coloured thick pus.

*Liver, spleen, kidney.*—Nothing of note.

*Intestine.*—Marked congestion and superficial erosion for 20 cm. above the ileo-caecal valve.

*Colon.*—Slightly thickened and much ulcerated, particularly at caecum, and at hepatic and splenic flexures, and in the terminal 30 inches.

Microscopical examination of contents of pulmonary abscess and large intestine revealed numerous typical living *Entamoeba histolytica.* Culture of mucus from surface of colon gave an agglutinating Flexner type of dysentery bacillus.

H. M. H.

HALL (G. A. M.). **A Case of Amoebiasis of the Lung.**—*China Med. Jl.* 1926. July. Vol. 40. No. 7. pp. 644–646. [7 refs.]

The author disclaims any considerable scientific value for his report of this case. His object is to draw attention, in China, to the existence of such cases—that they may the more readily be diagnosed, and an easily curable condition saved weeks of unnecessary invalidism.

A Chinaman, of 42 years, with fever, cough, anchovy sputum, no hepatic tenderness, dysentery denied. Physical signs denoted lesion of lung below angle of right scapula. 20 cc. of thin greenish albuminoid fluid aspirated from this area ; content predominantly polymorphonuclear ; no amoeba found, but material was not examined fresh. Repeated examination of sputa revealed no tubercle bacilli ; no fresh sputum smears were examined for amoebae. Stools well formed—no blood or mucus and no amoebae or cysts found on repeated examinations. Sigmoidoscope showed smooth and perfectly healthy mucosa. On 23rd day ½ gr. emetine hydrochloride was given intramuscularly and repeated every morning. Rapid improvement followed. After 12 doses, patient felt perfectly well and all symptoms and physical signs had disappeared.

After a brief survey of some of the recorded evidence in favour of primary amoebic abscess of lung, the author notes that in a large number of these cases, as in the one he reports here, diagnosis was made only when the condition was found to clear up rapidly under emetine, which LOISELET describes as " the diagnostic and therapeutic ' touchstone '." [The use of emetine in pulmonary inflammations with blood-stained sputa has been followed by disappearance of symptoms and physical signs ; so much has often been reported. The same had long been known of vinum ipecac. That the efficacy of emetine in these cases has diagnostic value may indeed be true ; but it does not rest, so far, on any exact observation or critical analysis.]

H. M. H.

PAWAN (J. L.). **A Case of Pyelitis associated with** *Entamoeba histolytica.* —*Ann. Trop. Med. & Parasit.* 1926. June 24. Vol. 20. No. 2. pp. 199–200.

The author notes the statement of DOBELL, that BAELZ (1883) was the first to describe *E. histolytica* as occurring in the urine. CASTELLANI

and CHALMERS recognize amoebic pyelitis as a definite clinical and pathological entity.

The case here reported was that of a woman, aet. 58 years, who had had periodic attacks of dysentery with blood, mucus and *E. histolytica* in stools, relieved by emetine injections. Last attack end of November, 1925; December 27th following she presented signs of acute cystitis (no vaginitis), clearing up with rest and urotropine. On January 6th following she developed acute pain and tenderness in left loin, rigors and T. 104·8° F. Urotropine and alkaline diuretics gave only slight relief. On January 12th microscopic examination of urine revealed pus, bladder and renal cells, and vegetative forms of *E. histolytica*. On 13th catheter specimens of urine, carefully taken, also showed *E. histolytica*. Emetine injections were followed by rapid disappearance of all symptoms and a urine normal on repeated examinations.

H. M. H.

PARDO (J. M.). [**Amebic Dysentery in Children.**]—*Medicina Ibera.* Madrid. 1926. Feb. 27. Vol. 20. p. 245. [Summarized in *Jl. Amer. Med. Assoc.* 1926. July 31. Vol. 87. No. 5. p. 368.]

A nine months old infant, belonging to a healthy Madrid family living in the best conditions, developed dysentery, amoebae being found in the stools. A watchmaker's child had dysentery and amoebic cysts were later found in its stools. The nurse of infant No. 1 had been having a watch repaired; watchmaker and nurse had apparently acted as intermediate carriers of the amoeba. Cure was realized in both cases with emetine—never more than 0·005 gm. per kilogram.

H. M. H.

TANON (L.) & TRABAUD. Cholécystites amibiennes primitives. [**Primitive Amoebic Cholecystitis.**]—*Rev. Méd. et Hyg. Trop.* 1926. May–June. Vol. 18. No. 3. pp. 65–73.

Three cases with symptom complex of cholecystitis. One gave a history of previous diarrhoea and a few active amoebae were found in the stools. In neither of the others was there history of dysentery, nor were amoebae demonstrated in any excretion. In none was there any sign of concomitant hepatic involvement. The diagnosis of amoebic lesion of the gall bladder was based on the pretext that, in fact, disappearance of symptoms and physical signs followed on emetine therapy [a flimsy pretext].

H. M. H.

REHBEIN (Max). Praktische Erfahrungen mit der Yatrenbehandlung der Amöbenruhr. [**Practical Experience of the Yatren Treatment of Amoebic Dysentery.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1926. July 1. Vol. 30. No. 7. pp. 294–297.

The author's experience of yatren treatment in over 200 cases of acute and chronic amoebic dysentery occurring in Barranquilla, Colombia, leads him to the emphatic statement that for all forms of this dysentery yatren surpasses other known remedies, such as E.B.I., salvarsan and stovarsol.

H. M. H.

MÜHLENS (P.). Cinco años de tratamiento de la disenteria amibiana por el "Yatren 105." [**Five Years of Treatment of Amoebic Dysentery with "Yatren 105."**] —*Rev. Méd. de Yucatán.* 1926. June. Vol. 14. No. 1. pp. 13–14.

Professor Mühlens reports further excellent results from the use of Yatren 105 in amoebic dysentery. Chronic and relapsing cases, even of ten years' duration, rapidly cleared up with doses of 1 gm. thrice daily by mouth. In acute cases the cure was so rapid that the treatment might almost be regarded as abortive in its action. For very chronic cases, rectal administration of 1–3 per cent. solution was combined with pills by mouth. In some instances of bacillary dysentery also cure was effected by this drug.

H. Harold Scott.

DALMEYER (J. J.). "Yatren 105" bei Amöbenruhr. [**Yatren in Amoebic Dysentery.** — *Arch. f. Schiffs- u. Trop.-Hyg.* 1926. Oct. 1. Vol. 30. No. 10. pp. 654–656.

The author's experience of emetine and emetine-bismuth-iodide in the treatment of amoebic dysentery in Java had shown the necessity of admitting the patients to hospital for treatment, and had been disappointing in that relapse and subsequent chronicity of dysentery were not rare. In four years' experience yatren has been unfailingly successful, both in acute and chronic and "larval" amoebic dysentery. Stool examinations two and three years after treatment have revealed no relapses. It was not always necessary to admit the patients to hospital for treatment. A course of treatment lasted usually only seven days. Yatren was given *per os* 1 gramme three times a day; some required only ½ gm. t.i.d. Children tolerate it well.

In one case of undoubted post-dysenteric liver abscess yatren brought about cure without operation.

The author concludes with the hope that the advent of yatren will be followed in the near future by the disappearance of sprue in so far as this disease is caused by amoebic dysentery.

H. M. H.

TURNER (R. H.) & JONES (P. H.). **Yatren 105 in the Treatment of Amebic Dysentery. A Preliminary Note.** —*New Orleans Med. & Surg. Jl.* 1926. Oct. Vol. 79. No. 4. pp. 233–237. [6 refs.]

A preliminary report on eight cases of amoebic dysentery. Five cases were "in first attack"; four of them had had symptoms for from ten days to one year, and one for 12 years. Three cases had had previous attacks in from one to 15 years. One of these had been treated many times very thoroughly with ipecac, emetine and stovarsol. Seven showed ulcers in rectum. In all typical vegetative forms of *E. dysenteriae* (*histolytica*), motile and containing red blood cells, were found.

Yatren was given 1 gramme three times a day for first and third weeks—during second week no medication. Only two cases were kept in bed and on soft diet. It was noted that during first week of treatment acute symptoms were relieved and ulcers showed rapid healing. All gained weight, with improved appetite and sense of well-being. Iodism or other unpleasant symptom did not develop. No proof of

permanence of cure of these cases can yet be given; five cases, followed one month and more after treatment, have shown no clinical or microscopic evidence of acute or chronic amoebiasis.

H. M. H.

AKASHI (Shinryu). [**On the Therapeutic Efficacy of Yatren against Amoebic Dysentery.**]—*Tokyo Iji-Shinshi* (*Tokyo Med. News*). 1925. Nov. No. 2448. [Summarized in *Japan Med. World.* 1926. Feb. 15. Vol. 6. No. 2. p. 43.]

The author "proved" that Yatren kills the amoebae infesting the intestinal lumen and then gradually attacks those in the superficial layer of the intestinal wall and in the ulcers. The killing of the amoeba proceeded first of all by destruction of its protoplasm.

Emetine, on the contrary, did not act on amoebae in lumen of intestine, but against the large forms in the gut tissues. Amoebic dysentery therapy would therefore be most effective by the use of these two drugs.

[The abstract (paraphrased above) gives no indication of the data on which these interesting and clinically important conclusions are based.]

H. M. H.

YOUNG (W. A.) & TUDHOPE (G. R.). **The Pathology of Prolonged Emetin Administration.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1926. Mar. 18 & May 20. Vol. 20. Nos. 1 & 2. pp. 93–99. [2 refs.]

One of the authors suffered from severe *E. histolytica* dysentery. The amoebae persisting, 18 grains of emetine hydrochloride were given, 1½ grains per diem, 1 grain intravenously and the half-grain subcutaneously. About 14 days after this course of injections he noticed weakness of arms and legs, ankle drop and some muscular wasting. Extensors and flexors were equally affected. No pain subjectively and objectively, none on squeezing muscles and deep pressure on sciatic nerve and its branches. Knee jerks sluggish. The only sensory symptom observed was loss of taste for tobacco and food. Eye accommodation quickly became fatigued. This syndrome—emetine palsy—lasted for one month. WALTERS and KOCH (1917) describe such symptoms, but the authors could find no account of the pathology of the condition. Text books refer to symptoms of neuritis; this is a misnomer as applied to the direct action of emetine, for by neuritis is meant inflammation of nerve trunk. Their experiments on rabbit and guineapig injected intraperitoneally with emetine revealed that this does not occur. They observed the pharmacological effects of emetine described by PELLINI and WALLACE (1916), except that these workers, with larger doses, found ulcers in stomach and gut. Three of the authors' emetinized animals developed distinct weakness of hind legs.

Post-mortem examination of the injected animals revealed general loss of fat; dilated and injected heart; emphysema and injection of lungs; liver and spleen enlarged and hyperaemic; kidneys enlarged with haemorrhages on surface; stomach and intestines injected only, and albuminous urine in bladder.

*Histology.*—Heart muscle.—Hyperaemia, granular and cloudy swelling, nuclear staining poor, haemorrhages between fibres, no fatty change. Lungs.—Hyperaemia, emphysema, alveolar walls thickened. Spleen.—Hyperaemia only. Liver.—Dilated capillaries, granular and cloudy change, necrotic areas, nuclear staining poor. Kidneys.—Hyperaemia, tufts swollen, desquamation of epithelium, cells swollen and granular,

haemorrhagic and necrotic area, casts. Suprarenals.—Congestion and haemorrhages in medulla. Muscle.—Granular and cloudy changes. Peripheral nerves.—Wallerian degeneration at distal end. Spinal cord.—Anterior motor horn cells, chromatolysis. No evidence of sensory fibre degeneration.

The findings harmonized with the varying dosage of emetine. With the bigger doses, the process was of an acute nature; with medium doses pathological changes were more marked, more time having been allowed for their production. With less doses it took longer time to produce the same changes, indicating a cumulative action of emetine. This is supported by the fact that palsy may develop some considerable time after cessation of emetine administration. [WALTERS and KOCH demonstrated pharmacologically its cumulative action.] When emetine is given continuously, the safety threshold for 4 lbs. rabbit is less than $\frac{1}{45}$ grain. On this basis for a 12 stone man, eleven doses of 1·9 grain would kill him and 35 doses of 0·8 grain would be distinctly risky. In fact, experience has already shown that more than one grain in 24 hours should not be given, and not more than 12 grains in all. The pathological picture shows that emetine is essentially a protoplasmic poison irrespective of type of tissue. The changes in heart muscle alone would be sufficient to kill. There was no inflammation whatsoever of nerve or its sheath. A motor fibre degeneration as in lead palsy is a possibility. It is doubtful whether recovery of the animal could occur if nerve degeneration were reached, for by then other organs show too advanced pathological changes. Weakness after emetine treatment is due to direct action of emetine on the muscle protoplasm. Exercise is therefore contra-indicated since the heart muscle must also be similarly affected. In cases where heart or kidney is affected, it is advisable to give as small a dose as is possible for the treatment of the dysentery.

[This investigation emphasizes, once more, the importance of accurate diagnosis before treatment of dysentery. The life of a toxic case of bacillary dysentery must be gravely jeopardized by emetine therapy—even by a few "diagnostic" doses.]

H. M. H.

NEWMAN (R. E. U.) & DAVIES (R. M.). **A Comparison of Stovarsol and Emetine Hydrochloride in the Treatment of Amoebic Dysentery.**—*Jl. Roy. Army Med. Corps.* 1926. Oct. Vol. 47. No. 4. pp. 296–300.

Eighteen cases of dysentery at Quetta, in none of which was there history of dysentery in the previous two years. In stools of all, actively motile amoebae with ingested red blood corpuscles were found. Alternate cases were treated with emetine and stovarsol. (*a*) Emetine hydrochloride 1½ gr. in one hypodermic injection daily for 12 days—a total of 18 gr. (*b*) Stovarsol—8 gr. daily for 10 days—a total of 80 gr. All the patients were kept in bed on milk diet, saline aperient in early morning, and Deeks' bismuth treatment—2 drachms bismuth carbonate in half a glass of soda water three times daily. The object of this is to render the acid contents of the colon alkaline, and to increase alkalinity of the portal blood stream. The alkalinity is at its height 2½ hours after administration of each dose of bismuth, and it is then, according to ACTON and KNOWLES, that the emetine should be given. Stools were examined for cysts on six definite occasions after completion of treatment, i.e., 4th, 11th, 18th, 19th, 20th, 21st days. The results are

tabulated. Although, as the authors point out, the number of cases is small, yet the results show that stovarsol is nearly as efficacious as emetine hydrochloride. Except for one case of erythema, stovarsol produced no ill after-effects. In allaying the tenesmus and acute abdominal pain at the onset of the illness emetine hydrochloride was the more effective. The danger of cardiac damage must not be overlooked; the authors believe that a course of emetine should not exceed 12 to 15 gr. It is possible that a combination of the two drugs may eventually prove the best method.

H. M. H.

BROWN (P. W.). **Treatment of Endamebiasis: Stovarsol.**]—*Illinois Med. Jl.* Oak Park. 1926. May. Vol. 49. p. 410. [Summarized in *Jl. Amer. Med. Assoc.* 1926. July 10. Vol. 87. No. 2. p. 126.]

Emphasis is laid on the wide distribution of *E. histolytica*. Emetine is invaluable and is particularly effective in rapidly controlling most of the acute phases of the infection. The organic arsenical compounds seem to be more effective in eradicating the parasite than are ipecac and its derivatives. Cure depends more on persisting with varying treatments than on any one drug. From a small series of cases of ulcerative amoebic proctitis the indications are that recurrence is more likely in this group than in those without gross ulcerative lesions.

H. M. H.

i. YORKE (Warrington) & ADAMS (A. R. D.). **Observations on *Entamoeba histolytica*. I. Development of Cysts, Excystation, and Development of Excysted Amoebae *in Vitro*.**—*Ann. Trop. Med. & Parasit.* 1926. Aug. 13. Vol. 20. No. 3. pp. 279–302. With 4 plates. [14 refs.]

ii. —— & ——. **II. Longevity of the Cysts *in Vitro*, and their Resistance to Heat and to Various Drugs and Chemicals.**—*Ibid.* pp. 317–326. [9 refs.]

i. It has been generally accepted, the authors state, that cysts of *E. histolytica* will hatch only after subjection to action of gastric and/or pancreatic juices. This hypothesis is probably based on observations of UJIHARA (1914), PENFOLD, WOODCOCK and DREW (1916), CHATTON (1917), and CUTLER (1919). The work of DARLING (1913) had been apparently ignored. DARLING described gradual disappearance of cysts from moist chamber preparations of heavily infected faeces, development of amoebulae within the cysts, and their emergence [this Bulletin, Vol. 1, p. 462]. DOBELL and O'CONNOR (1921) state that "it is certain that the cysts never hatch in the colon, where they are found, or outside the body." SELLARDS and THEILER (1924) showed that *E. histolytica* cysts would hatch when injected intrarectally in kittens. Their very carefully controlled work has been confirmed by HOARE (1925). This reopens the whole subject regarding conditions necessary for excystation of *E. histolytica* and possibly has a bearing on the factors concerned in amoebic dysentery relapse. With the object of throwing further light on the matter the authors have conducted a large number of experiments which are fully described in this paper.

1. They find that *E. histolytica* cysts develop and excyst under suitable conditions *in vitro*.

2. Moisture and a suitable temperature (about 37° C.) are essential for excystation. Passage of cysts through *liquor pepticus* or *liquor pancreaticus* is unnecessary for excystation.

3. Development and excystation can readily be followed in cultures of *E. histolytica* cysts on Locke-egg-serum, and certain other media at 37° C.

4. Great variation in the stage of development of *E. histolytica* cysts may be seen in the freshly-passed faeces of different individuals, and of the same individual at different times. These variations concern chiefly the number of nuclei, the glycogen content, and the percentage containing chromatoid bodies.

5. The youngest cysts are uninucleate and loaded with glycogen; during development chromatoid bodies appear, the nuclei divide, and glycogen decreases. Later, the cyst becomes quadrinucleate; chromatoid bodies are well developed, but glycogen is much less evident or entirely absent. When the cyst is mature and ready to excyst, the nuclei are agglomerated and the cytoplasm is homogeneous and without glycogen; the chromatoid bodies are greatly reduced or absent.

6. They failed to observe any evidence of autogamy in the development of the cysts, but frequently saw chromatin particles apparently extruded from the nuclei immediately prior to, or during, division.

7. The freshly excysted *E. histolytica* contains four closely agglomerated nuclei, and the cytoplasm is very finely-alveolar. It moves in characteristic slug-like manner, with nuclei almost invariably anterior, and drags behind a more or less motionless tail; as bacteria are ingested the cytoplasm becomes more alveolar and digestive vacuoles quickly appear.

8. In cultures the majority of the quadrinucleate excysted amoebae divide, either directly or indirectly, into four uninucleate individuals, but in a certain proportion the cytoplasm fails to divide and multinucleate individuals are formed. Here again no indication of autogamy was observed.

9. They describe a method of obtaining concentrated washed suspensions of *E. histolytica* cysts. Inoculation on Locke-egg-serum or other suitable medium with such suspensions is the simplest way of obtaining excellent cultures of vegetative forms.

ii. The usually accepted method of determining the vitality of cysts of *E. histolytica* has been that of KUENEN and SWELLENGREBEL (1913): dead cysts stain with weak eosin-water solutions, and living cysts do not. It has been impossible to decide whether this is wholly true. Inoculation of kittens means at least a great sacrifice of animals. The longevity of the cysts is, from the public health point of view, a matter of fundamental importance. Records of various workers indicate that cysts live longer in water than in faeces, and at low temperatures than at higher or at body temperature. Under favourable conditions cysts have been stated to survive for 29 days (KUENEN and SWELLENGREBEL), for over 30 days (WENYON and O'CONNOR), up to five weeks (DOBELL), 153 and 211 days (BOECK).

The authors describe their experiments. Their test for longevity of cysts is based on the fact that they can readily be cultured *in vitro*. They find that :—

1. The eosin reaction is not a reliable criterion of viability of *E. histolytica* cysts. Those which stain with dilute eosin are dead, but not all those which do not stain are alive.

2. *E. histolytica* cysts begin to die fairly rapidly in faeces kept at laboratory temperature (16° to 20° C.) for 3 or 4 days, and all are dead within about 10 days ; approximately the same result is obtained when the faeces is kept at 0° C. in the ice chest.

3. Washed suspensions of *E. histolytica* cysts in water live longer, especially if kept at 0° C., but even under these conditions live cysts are not found after three weeks.

4. *E. histolytica* cysts survive a temperature of 45° C. for 30 minutes, but are killed within five minutes by a temperature of 50° C.

5. *E. histolytica* cysts are remarkably resistant to emetine and to yatren, and relatively so to hydrochloric acid and chlorine. The last-named in strengths far in excess of that used in the bacteriological sterilization of water has no effect on the cysts.

6. The following chemicals were found to be lethal for the cysts in the strengths as stated, both at 37° C. and at laboratory temperature: $HgCl_2$ 1 in 2,500 ; formaldehyde, 0·5 per cent. ; carbolic acid, 1 in 100 ; lysol, 1 in 100 ; "Milton," 2·5 per cent. ; pot. permang., no effect at 1 in 100 ; yatren, no effect in 5 per cent. solution ; emetine HCl, no effect in 5 per cent. solution.

H. M. H.

THOMSON (Margaret Dora). **Experimental Amoebiasis in the Rabbit.**—*Univ. California Public. Zool.* 1926. June 23. Vol. 29. No. 2. 23 pp. With 5 text figs. [24 refs.]

Carnivores have been found to be, apparently, the most suitable animals for obtaining experimental infections of *E. dysenteriae*. LÖSCH (1875) infected a dog, but by far the greatest amount of experimental work has been performed with cats which, like the dog, acquire a fatal dysentery. Rodents have appeared to be rather refractory to infection by *E. dysenteriae*. The author reviews and discusses the attempts (some reported successful) of various workers with rat, mouse, guineapig, rabbit and gerbil, and reports fully her own experiments.

Motile amoebae in bloody mucous stools of infected cats failed to infect a half-grown rabbit when rectally inoculated. Of three rabbits fed mature cysts of *E. dysenteriae* obtained from chronic human cases of amoebiasis one acquired an infection and died 30 days after the initial feeding. No cysts were ever found in the stools during experimentation, nor at autopsy in the intestine of the infected rabbit. The infection was localized in the caecum. Microscopical examination of sections of the caecal wall showed an amoebic invasion of the mucosa, sub-mucosa and the circular muscular layer. Lymph nodes and channels were heavily infected. Amoebae were also found in the lumen of the blood vessels. [The text figures illustrate, in so far as microphotographs can do so, this invasion of the tissues of the gut.] Five dogs were fed human faeces containing mature cysts of *E. dysenteriae*. No infection resulted. Unsuccessful attempts were made to produce an infection in two young kittens with cysts from an infected rat and later with cysts from chronic human cases. A few months afterwards one of them was infected by rectal inoculation with dysenteric stools from cats with acute amoebiasis.

H. M. H.

CARVAILLO (R.) & SAUTET (J.). Recherches expérimentales sur quelques localisations extra-intestinales de l'amibose. **Extra-Intestinal Infections with Amoebae: Experimental.** —*Ann. Parasit. Humaine et Comparée.* 1926. Oct. 1. Vol. 4. No. 4. pp. 349–351, [10 refs.]

Recent observations, especially those of PETZETAKIS and Madame PANAYOTATOU, on urinary and pulmonary amoebiasis, induced the authors to attempt to reproduce experimentally these amoebic manifestations. They employed cultures of *Entamoeba dysenteriae* (made by BRUMPT, from a patient with amoebic dysentery, which had not responded to the usual treatment). This culture, after the 78th passage (at the commencement of their experiments) killed a kitten to which it had been intrarectally inoculated,

1. The external urinary meatus in seven kittens was sealed by collodion tampon. Median supra-pubic cystotomy was performed and the bladder inoculated with the amoebae. After two hours the meatus was unsealed. (Inoculation by catheterization of bladder was not done on account of the narrowness and fragility of the kitten's urethra.)

Three kittens died in the first week from peritonitis and infiltration of urine. At autopsy one only showed a 3 mm. ulceration of the bladder mucous membrane; sections of this ulcer (haemalum and eosin stained) revealed no amoebae. No other sort of lesion in bladder was found in any of the three. The authors note that in face of this, the finding of "dysenteriform" ulcers by cystoscopy does not suffice for a diagnosis of amoebiasis. The four other kittens tolerated the operation and were killed after ten days. A daily examination of their urine had revealed no amoebae. At autopsy none showed any lesion of the bladder. On an eighth kitten cystostomy was performed. This permitted two successive inoculations of the bladder with the amoebae, and repeated examinations of the urine. At autopsy no lesion of bladder was found.

2. BRUMPT inoculated kittens with the amoebae by tracheal puncture. At autopsy no lesion was found. The authors repeated this experiment with one kitten—at autopsy no macroscopic or microscopic lesion of the pulmonary passages or mucous membranes, nor amoebae, could be found.

3. Pleural inoculation provoked purulent effusion which killed the kitten in 24 hours. Examination of the pleural effusion and sections of lung and pleura (haemalum-eosin stained) revealed no amoebae. BRUMPT had inoculated kittens intrapleurally and intraperitoneally with cultures (different from that used by the authors) of *E. dysenteriae* and Trichomonas. With the amoebae the result was negative, but Trichomonas survived perfectly.

[Now the kitten is peculiarly susceptible to infection with the amoeba of dysentery, and many will agree with the authors that the proofs for the amoebic nature of most of the reported human urinary and pulmonary cases are not entirely conclusive.]

H. M. H.

CRAIG (Charles F.). **A Simplified Method for the Cultivation of** *Endamoeba histolytica.*—*Amer. Jl. Trop. Med.* 1926. Sept. Vol. 6. No. 5. pp. 333–339. [4 refs.]

(1). Locke solution containing one part to seven of inactivated human, horse or rabbit blood serum was found very efficient in the

cultivation of *E. histolytica*. Best results were obtained when inactivated human blood serum was used.

(2). Excellent growth of *E. histolytica* in this medium demonstrates that coagulated egg or blood agar slants, used as a substratum, are not essential for growth of this species; and that neither egg nor blood is essential as food supply.

(3). In this medium *E. histolytica* remained alive and motile for 11 days, and successful transfers were obtained after eight days. Best results were obtained by making transfers every 24 or 48 hours.

(4). Transfers of *E. histolytica* in this medium made at intervals of 24 and 48 hours were continued for over three months, during which time the amoebae did not change in morphology or in reproductive ability.

H. M. H.

SAUTET (J.). Action de l'amidon sur les cultures d'amibes. [**Action of Starch on Cultures of Amoebae.**]—*Ann. Parasit. Humaine et Comparée.* 1926. Oct. 1. Vol. 4. No. 4. pp. 345–348. [10 refs.]

The author describes his experiments and technique. He finds that rice starch grains, sterilized or not, enrich cultures of amoebae and prolong their life. Other starches, e.g. arrowroot, wheat, potato, manioc, chestnut, appear to have no effect on the cultures. Drbohlav's starch medium, in six cases, gave better results than Dorset medium for isolating amoebae.

H. M. H.

ST. JOHN (J. H.). **Differential Characteristics of the Amoebae of Man in Culture.**—*Amer. Jl. Trop. Med.* 1926. Sept. Vol. 6. No. 5. pp. 319–331. With 22 figs. on 3 plates. [2 refs.]

The method of culture for detection of Entamoebae has obvious advantages over the usual hit-or-miss procedures. Were it not for the involved technique incident to staining, it is believed that more clinicians would become interested in attempting to differentiate the amoebae. By culture on Locke-egg serum media (BOECK and DRBOHLAV) a ready technique is at hand for the study of the different characteristics of various species of amoebae. The author's interesting paper shows that in culture the definition of the visible structure and observation of the movements peculiar to *E. histolytica*, *E. coli*, *E. nana*, *Iodamoeba williamsi*, *Dientamoeba fragilis* give valuable aid in differentiating these amoebae (found in man). He found no remarkable change in structural detail or behaviour. The main identifying features conform closely to the accepted descriptions. In culture, however, the unlimited number of amoebae available for study permits of greater emphasis being placed on minor individual peculiarities. A full description of these features and peculiarities is given in the paper.

As addendum he gives the results of a single examination by method of culture of stools of 73 members of the class at the Army Medical School.

| | | | |
|---|---|---|---|
| *Positive.* | Flagellates | 5 | 23 or 31 per cent. |
| | Amoebae | 11 | |
| | Both | 7 | |

*Amoebae species found.*—*E. histolytica* 7–9·6 per cent.; *E. coli* 5–6·8; *D. fragilis* 1–1·3; *I. williamsi* 1–1·3; and *E. nana* 4–5·9 per cent. *Flagellate species found.*—Chilomastix 6–8·2 per cent.; Trichomonas 5–6·8; Pentatrichomonas 1–1·3; Tricercomonas 3–4 per cent.

H. M. H.

KESSEL (John F.) **Some Similarities between the Dysentery Amoeba of the Monkey and of Man.**—*Proc. Soc. Experim. Biol. & Med.* 1926. May. Vol. 23. No. 8. pp. 675–676. [2 refs.]

The dysentery amoeba of the monkey and that of man are so much alike in structure as to have evoked a suggestion of their identity. The author, by means of cysts, succeeded in infecting two clean monkeys with *E. histolytica* from man, and from the faeces of these monkeys he subsequently infected two clean cats, in whose gut the typical lesions of amoebic dysentery were demonstrated; and he offers the results of these experiments as reasonable evidence of the specific unity of the two stocks—from man and monkey respectively.

[On the other hand, Das GUPTA, in India, was unsuccessful in attempts to infect kittens with *Entamoeba nuttalli* obtained by culture from a moribund monkey, *Macacus rhesus*.]

A. Alcock.

KULO (R.). **Observations on** *Dientamoeba fragilis*.—*Amer. Jl. Trop. Med.* 1926. July. Vol. 6. No. 4. pp. 299–305. With 21 figs. on 1 plate. [10 refs.]

These observations were made on material from a hospital patient in Illinois who had been suffering for some time from dysentery suspected to be amoebic. The organism specified in the title was found in enormous numbers in stools (along with small numbers of *Chilomastix mesnili*, *Entamoeba coli* and *E. nana*).

Some description is given of *Dientamoeba fragilis* and of its nucleus in division. Binucleate individuals were found in only 12·2 per cent. of 2,000 observed at random.

A. A.

HOARE (C. A.). **Sections of the Intestine of a Kitten presumably infected with** *Entamoeba histolytica* **by Rectal Injection of Cysts alone. [Laboratory Meeting Demonstration.]**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1925. Nov. 19 & Dec. 17. Vol. 19. Nos. 5 & 6. pp. 277–278.

The view had been generally held that *E. histolytica* cysts were incapable of liberating the enclosed amoebae until the cysts had been passed out of the body and ingested by another individual. SELLARDS and THEILER (1924) infected kittens by injecting into the large intestine material containing cysts alone. The author obtained faeces from the London Hospital, which on examination, when 24 hours old, revealed cysts only. A portion was emulsified in tap water, allowed to settle for half an hour, and centrifuged. This procedure was repeated twice. Several examinations of the faecal sediment revealed cysts only. 5 cc. of the emulsion was injected into a kitten per rectum, and its anus was sealed by cotton-wool-collodion-plug. The plug was removed four days later, when examination of faeces revealed no amoebae. Three

weeks after inoculation the kitten passed blood and mucus for three days, during which time stools revealed no amoebae. The stools then became normal. The kitten was killed 41 days after inoculation. Macroscopically the large intestine appeared normal and without ulceration. Microscopic examination of sections revealed amoebae concentrated in patches at base of the mucous membrane. The author notes that, although the possibility of individual active forms surviving in the inoculated material cannot be entirely excluded, yet as this material was 24 hours old, revealed cysts only, and was several times washed, before inoculation, it is highly probable that the infection in the kitten was produced by the cysts alone.

H. M. H.

BRUMPT (E.). Infection expérimentale du chat par l'*Entamoeba coli* Loesch, 1875 Schaudinn emendavit 1903. [**Experimental Infection of Cat with** *Entamoeba coli.*]—*Ann. Parasit. Humaine et Comparée.* 1926. July 1. Vol. 4. No. 3. pp. 272–285. With 6 text figs. [25 refs.]

The author gives a full account of his experiments. He reviews briefly previous attempts of various workers to infect the cat with *E. coli*. He himself had failed before using the method, discovered by BOECK and DRBOHLAV, of occlusion of the animal's anus. His successful infection of the cat, in this case, was due to the fact that an invagination of the small intestine had produced intestinal stasis. SELLARDS has clearly demonstrated that faecal stasis promotes experimental amoebic infection of the gut. *E. coli* has been held to be non-pathogenic for man. Theobald SMITH 1910 showed that the pig amoeba (*E. polecki* Prowazek 1912—*E. suis* Hartmann 1913) could multiply in the intestinal ulcerations of *la peste porcine.*

HAMMERSCHMIDT has described in four human cases, at autopsy, penetration of the gut wall by amoebae presenting the morphological character of *E. coli*. Other authors, whose findings are briefly mentioned, have more or less incriminated *E. coli* as a pathogenic agent in man. The author's experiments here detailed show that, perhaps in exceptional circumstances, *E. coli* can penetrate small ulcerations in the gut of a kitten which it has itself produced, or which were caused by *E. dispar* with which the *E. coli* was associated in this experiment. The author claims that this accidental histotropism of *E. coli* in the kitten, denotes that this amoeba can become pathogenic for man in certain favourable conditions.

[The photomicrograph accompanying the text shows what may well be amoebae in the gut tissues of the kitten, but fails to demonstrate them as *E. coli*.]

H. M. H.

i. PETRIDIS (Pavlos Ar.). Un cas d'abcès du foie traité sans succès par les ponctions répétées et les injections d'émétine et définitivement opéré. [**Liver Abscess—Aspiration—Emetine Injections Incision.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1926. July 1. 3rd Ser. Year 42. Vol. 50. No. 23. pp. 1140–1145.

ii. HARRISON (C. H.). [**Treatment of Chronic Amebic Dysentery and Ameba Carriers with Stovarsol.**]—*U.S. Veterans' Bureau Med. Bull.* Wash. 1926. May. Vol. 2. p. 447. [Summarized in *Jl. Amer. Med. Assoc.* 1926. July 17. Vol. 87. No. 3. p. 202.]

iii. PETRIDIS (Pavlos Ar.). Péritonite généralisée purulente consécutive à la rupture d'un abcès du foie de la partie postérieure de la face convexe du lobe droit et ayant simulé la péritonite par perforation intestinale typhoïdique. **Peritonitis following Rupture of Liver Abscess.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1926. July 1. 3rd Ser. Year 42. Vol. 50. No. 23. pp. 1145—1149.

iv. VIOLLE (Cesar). **Nuclear Division in the Trophozoites of** *Endamoeba histolytica.*—*Proc. Nat. Acad. Sci.* 1926. May. Vol. 12. No. 5. pp. 305–311. With 24 figs. on 1 plate. [4 refs.]

v. PAL (B. D.). **Paresis following Emetine Injections.**—*Indian Med. Gaz.* 1926. Oct. Vol. 61. No. 10. pp. 498–499.

i. The condition was a sequel to amoebic dysentery. Amelioration followed aspirations and emetine injections, but as the patient's condition still remained grave, incision was carried out and *two litres* of chocolate coloured pus were evacuated.

ii. Good results were obtained by stovarsol 0·5 gm. by mouth three times daily. Colonic irrigation with 4,000 to 8,000 cc. physiologic sodium chloride solution, followed by 1 in 500 mercurochrome, were routine procedures in these cases.

iii. At autopsy, scrapings of the abscess wall revealed entamoebae.

H. M. H.

iv. The author has been studying the phenomena of nuclear division as exhibited in stained specimens of trophozoites of *Entamoeba histolytica*, and he here describes and illustrates (in some instances by microphotography) the results. The paper is one for the discriminative nucleologist.

A. A.

v. The author records briefly four cases of paresis in adults after three, two, six, and an unstated number of 1 grain injections of emetine hydrochloride. [It does not appear whether any of them had received a previous course of emetine injections.]

A. G. B.

## BACILLARY DYSENTERY.

BREAKEY (S. F.). **A Case of Infection with** *Bacillus dysenteriae* **(Flexner) presenting Unusual Symptoms. With Bacteriological Notes** by CLAYTON (F. H. A.).—*Lancet.* 1926. Sept. 11. pp. 541–542. [3 refs.]

In a certain number of cases symptoms produced by Flexner bacillus are entirely different from classical symptoms of dysentery. SAVAGE and Bruce WHITE recorded an outbreak of "food poisoning" due to Flexner bacillus, and a similar series of cases with sudden onset has been reported by BAINBRIDGE and DUDFIELD. The case here reported finds an almost exact parallel in a case of Flexner infection described by MARSHALL in 1909.

A girl, aged seven years, apparently quite well up to onset of vomiting (once). That night 20 or 30 calls to stool, but very little passed and no blood or slime noticed. In morning "excited and strange in manner;" sent to hospital, where were found exaggerated knee jerks, dilated pupils, photophobia, temperature 102·6°, left lateral decubitus with flexion of thighs and legs. Later, complete unconsciousness and vomiting of small quantity bile stained fluid. Convergent squint left eye. A ribbon stool, greenish, no blood or mucus, passed. Later still, retraction of head, spasticity of limbs and positive Kernig. Lumbar puncture yielded clear fluid not under tension. Ever deepening coma terminating in death. Laboratory examination of C.S.F. revealed nothing pathological. Culture

from rectal swab showed Flexner dysentery bacillus, confirmed by testing against Flexner serum. At autopsy, naked eye inspection revealed nothing abnormal in intestines or appendix, nor in brain—no pus, turbid fluid or tubercles. There was some slight congestion in basilar region around pons.

H. M. H.

CHATTERJEE (Sisir C.). **A Case of Bacillary Dysentery.**—*Indian Med. Gaz.* 1926. Aug. Vol. 61. No. 8. pp. 393–394.

The diagnosis of bacillary dysentery in a woman of 30 at Nagpur was arrived at by the presence of: (1) High fever; (2) Blood and slime in stools, and (3) Failure of emetine and castor oil (administered by the author's assistant.).

Saline bowel washes were followed by subsidence of fever, and total disappearance of all symptoms occurred after a second dose of anti-dysenteric serum given intraveneously. The author remarks that bacillary dysentery is not common in Nagpur.

The editor, *Indian Medical Gazette*, appends a note as follows:—

"There is an almost universal habit in India of administering emetine in almost all cases of dysentery, irrespective of their causation. If the stools of Nagpur dysentery cases were examined bacteriologically, it would be found that what holds good for Calcutta, for most of India, holds good for Nagpur—that bacillary dysentery is some five or six times as common as is amoebic dysentery."

H. M. H.

SAISAWA (Kozo) & TANABE (Bunshiro). **Notes on Healthy Carriers of Dysentery Bacilli.**—*Philippine Jl. Sci.* 1926. July. Vol. 30. No. 3. pp. 335–346.

A valuable, interesting and thorough investigation at Tokyo of which full details are given.

(1). Among 2,847 recruits for the Japanese army, the authors found 15 (0·52 per cent.) healthy carriers of dysentery bacillus. Apparently healthy carriers were also found among the soldiers living in barracks in Tokyo. Out of 4,648 examined, 15 (0·32 per cent.) were found to harbour the dysentery bacillus.

(2). From the total of 30 carriers they isolated Y (Hiss-Russell) type in 17; Flexner in 10; nonfermenter of mannite in 2; and typical Shiga in only one case.

(3). In the majority of the carriers longer or shorter periods of positive culture alternated with periods of negative culture—negative periods were sometimes as long as four weeks.

(4). The carrier state persisted in some for as long as 13 weeks after detection.

(5). Sigmoidoscopy revealed, in most of the cases, ulceration or inflammation of the lower part of sigmoid flexure and of the rectal ampulla.

(6). Sigmoidoscopy enabled the authors to obtain more regular results than did stool cultures. In cultures made from ulcers, mucus, or pseudomembrane, colonies, sometimes almost pure, of the dysentery bacilllus were obtained without difficulty.

(7). The authors conclude that the great majority of so-called healthy or contact dysentery carriers are nothing but very mild cases of dysentery with such slight pathological and clinical manifestations that an infection by *B. dysenteriae* is not suspected.

H. M. H.

ELKELES (Gerhard) & SCHNEIDER (Alice). Zur Bakteriologie der Ruhr, besonders der diesjährigen [**Bacteriology of Dysentery.**]—*Deut. Med. Woch.* 1926. Aug. 20. Vol. 52. No. 34. p. 1424. [5 refs.]

The well known difficulty of a definite bacteriological specification in acute dysentery is emphasized. This has reference, of course, particularly to the slightly toxic or pseudodysenteric bacilli as distinguished from the well-defined Shiga-Kruse bacilli. The former have been divided up into a series of races numbered alphabetically, with the numbering so far reaching from A to J. The authors advise the practitioner to regard every case of typical acute clinical dysentery as "infective," that is to say bacillary dysentery, to report it as such, and to despatch the stool for laboratory examination as soon as passed.

W. F. Harvey.

SCHÖBL (Otto) & VILLAAMIL (Rita). **Contribution to the Serologic Grouping of *Bacillus dysenteriae* based upon the Quality of Antigen and Normal Agglutinins.**—*Philippine Jl. Sci.* 1926. May. Vol. 30. No. 1. pp. 1–38. [7 refs.]

The results of tests with one strain each of *B. dysenteriae* Shiga and Flexner showed that normal sera of horse, mule, sheep and goat agglutinated both in diminishing degree and the sera of guineapig and monkey agglutinated neither. Rabbit serum was peculiar, as it agglutinated the Flexner but not the Shiga type. This peculiarity was investigated for a large number of strains of the two types, and in addition, the effect of boiling upon the spontaneous agglutinability of suspensions. All the Flexner strains were agglutinable by normal rabbit serum but only one showed agglutination upon boiling for one hour. Of the Shiga series more than half were inagglutinable by rabbit serum. The inagglutinable strains showed no agglutination upon boiling. The agglutinable Shiga strains gave only a loose agglutinate and were divisible into a larger sub-group of members, which agglutinate spontaneously on boiling, and a smaller which do not. Flexner types of organism gave regular adsorption results, but the Shiga types were irregular in this respect. All degrees of agglutinability were found in these Shiga types in respect of normal rabbit serum, homologous immune horse serum, adsorption, simple boiling and acid; this is regarded as due to degeneration of antigen rather than to the presence of real agglutinin, such as is found for Flexner strains in normal rabbit serum.

W. F. H.

CHANNON (Hilda A.). **Identification of the Sonne Dysentery Bacillus in England.**—*Jl. Path. & Bact.* 1926. Oct. Vol. 29. No. 4. pp. 496–497. [5 refs.]

The identification of the Sonne bacillus here set out is complete. Examination of the faeces of a patient, with previous history of a

dysentery attack in Egypt, now suffering from diarrhoea, vomiting and wasting for three months, showed neither amoebae nor cysts. The lactose fermenting organism, isolated from the stools, agreed morphologically and in cultural reactions with a Sonne bacillus strain from the National Collection. The organism was pathogenic to rabbits: an agar slope suspension killed intraperitoneally within 12 hours. The Sonne bacillus occurs, according to CRUICKSHANK and KERRIN, rather commonly in Aberdeen, especially in outbreaks of dysentery in children. The serological tests of the isolated organism served to complete the identification. An antiserum prepared to this organism agglutinated both it and the Sonne bacillus at a titre of 1 in 640, while the Sonne bacillus antiserum agglutinated both organisms at 1 in 320. Complete cross absorption of agglutinins occurred with both sera. Neither antiserum agglutinated Flexner and Shiga bacilli.

W. F. H.

SARDJITO (M.). Onderzoek op de toxinevorming van Shiga-Kruse dysenteriebacillen in bouillon met verschillende waterstofionenconcentratie. [**Variation of Toxin Formation by Shiga-Kruse Bacilli with Hydrogen-ion Concentration.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1926. Vol. 66. No. 3. pp. 337–341. With 1 chart. [7 refs.]

The pH values of the test bouillons were 6·75, 7·1, 7·5 and 8·3; samples of 1 cc. were taken daily, diluted with distilled water to 3·5 cc., and tested colorimetrically for reaction by addition of 0·5 cc. of either 0·02 per cent. phenol-red or 0·04 per cent. brom thymol blue. In all five bouillons the reaction fell after the first day to near the turning point, after which the alkalinity rose, so that bouillon of initial reaction pH 7·5 reached 7·8 after the 10th day; all the other bouillons had reached this same point by the 17th day.

For the test of toxin formation, portions of each of the bouillons were filtered through a Berkefeld N candle on the 3rd, 7th, 14th and 21st day of culture and doses of 0·01, 0·02, 0·03, 0·04 and 0·05 cc. of the filtrate injected intravenously in white mice. The results for these cultures were: (1) Toxin formation did not begin till after the 3rd day; (2) toxin formation only began after a week in some; (3) The period of highest toxicity was between the 14th and 21st day; (4) the strongest toxin was obtained from the culture of initial reaction pH 7·5 whose filtrate after the 14th and 21st days was lethal in an intravenous dose of 0·01–0·02 cc.

W. F. H.

TADOKORO (Ryokichi) & SUGA (Kichinosuke). **Physiological Action of Dysentery Toxin.**—*Keio Igaku (Jl. Keio Med. Soc.).* 1925. July. Vol. 5. No. 7. [Summarized in *Japan Med. World.* 1926. Mar. 15. Vol. 6. No. 3. pp. 67–68.]

Perfusion of highly virulent toxin through isolated rabbit intestine increased peristalsis, and in larger doses caused paralysis. As, however, this action was exhibited after heating for one hour at 85° C., the authors conclude that it was due to other factors than the toxin itself. The clinical symptoms in dysentery, it is suggested, may be due to the demonstrable stimulation of the accessory sympathetic nerves of the large intestine, instead of to a non-specific hypersusceptibility arising from pathological change in the mucous membrane.

W. F. H.

CLIFFORD (Stewart H.). **Rôle of the Dysentery Bacillus in Arthritis Deformans (Type I). A Preliminary Report of Seven Cases, with a Note on the Intradermal Reaction in Dysentery.**—*Amer. Jl. Diseases of Children.* 1926. July. Vol. 32. No. 1. pp. 72–83. With 2 text figs. [10 refs.]

The first description of the arthritis following a definite clinical attack of dysentery is credited to SYDENHAM in 1672 following the dysentery epidemics of 1668–1672 in England. Since 1917 many observers have reported it. That the symptom complex called arthritis deformans of type I may, in some cases, be the arthritis following an unrecognized dysentery bacillus infection is the possibility raised by the observations reported in this paper. Seven cases of arthritis deformans are reported, in four of which there was evidence of there having been either an active or a past infection with *B. dysenteriae*, Flexner. The literature shows arthritis following a typical dysentery to be a well-established clinical entity that may occur as long as four weeks after the acute attack. It is possible to have a dysentery bacillus infection without its producing a clinical syndrome recognizable as dysentery. Dysentery should be considered as an aetiologic factor in arthritis deformans (type 1), and ruled out only after bacteriological studies and agglutination reactions, using a complete series of antigens, prove to be negative.

*Intradermal reaction.*—A formalinized vaccine of the organism isolated in one of the cases was used, 0·1 cc., being injected intradermally into all of the patients and their attendants; 24 hours later all showed oval, erythematous, raised diffused reactions. In every instance this pseudo-reaction had completely disappeared by end of the third day. In three of the four patients as the pseudo-reaction disappeared, the site of the original bleb became progressively deeper crimson and more indurated, until by the fourth day red indurated papules had been formed.

H. M. H.

BROKMAN (H.) & POPOWSKI (S.). **The Relative Frequency of the Positive Dysentery-Skin Reaction at Different Ages.**—*Jl. Immunology.* 1926. July. Vol. 12. No. 1. pp. 45–46. [3 refs.]

Brokman had previously determined the dependence of an intradermal skin reaction on the presence or absence of Shiga-Kruse antitoxin in the blood as in the Schick test for diphtheria. In the present study from the University Clinic for sick children in Warsaw, the authors use, as test dose, 1-100 of the lethal dose of toxin per kilogram of rabbit. Their results for 39, 262, and 374 persons examined at the age periods 0–3, 4–15, and 16–23, were 51, 27 and 59 per cent. of positive reactions.

W. F. H.

MULLER (L.). Milieux pour la recherche des bactéries du groupe typho-dysentérique. **Medium for Typho-Dysentery Organisms.**—*C. R. Soc. Biol.* 1926. Sept. 21. Vol. 95. No. 27. pp. 728–730. [2 refs.]

The medium is a simplification of one previously described.

Four solutions are required: No. 1, 40 gm. ferric pot. tartrate in 200 cc. distilled water, with the addition of 4 cc. liquid phenol; No. 2, 250 gm. lactose in 600 cc. distilled water; No. 3, 40 gm. crystalline pot. ferrocyanide

in 200 cc. distilled water; No. 4, Normal soda solution. These are well cooled. Only Nos. 2 and 3 require sterilization by heating for 30 min. at 100° C. Mix Nos. 1 and 2 solutions. Add 40 cc. of No. 4 solution and then No. 3 solution. This stock reagent mixture will remain sterile, even though frequently drawn upon for use. The actual medium is prepared by mixing 9 or 10 parts nutrient agar with 1 part of the stock reagent. Plates of this medium will keep for three weeks at room temperature and still longer in the ice chest.

*B. typhosus* colonies are orange-tinted on the brown medium, while *B. coli* colonies are blue.

W. F. H.

SARDJITO (M.). Immunisatie tegen bacillaire dysenterie door middel van bacteriënlysaat van Shiga-Kruse-bacillen, verkregen na inwerking met bacteriophaag. **Immunization against Bacillary Dysentery by Means of a Bacterial Lysate of Shiga-Kruse Bacillus, obtained by Action of the Bacteriophage.**—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1926. Vol. 66. No. 4. pp. 502–521. [12 refs.]

Sardjito gives an account of previous attempts of active immunization against bacillary dysentery. Vaccines prepared by KOLLE's method are too toxic. Admixture of anti-dysenteric serum diminished the toxicity but interfered with the degree of immunity which the vaccine is able to procure. BESREDKA's sensitized vaccine is also useful only to a certain extent. D'HERELLE's experiments offered the possibility of a new departure in this respect. In D'HERELLE's own experiments with Shiga-Kruse bacillus the lysate proved to be non-toxic whether administered *per os* or subcutaneously. The original toxins of the bacillus have been thoroughly altered, whilst at the same time the immunizing power of the lysate is much higher than that of the original strain of the bacillus. D'HERELLE ascribes the immunizing action of the lysate to the dissolved bacterial antigens, and not to a direct action of the bacteriophage itself.

In the author's own experiments two strains of bacteriophage were used in 73 rabbits. The lysate of Shiga-Kruse bacillus was still toxic, which Sardjito ascribes to a higher degree of virulence of the strain he used. It lost, however, its toxicity by treatment with formol, which left the immunizing power unaffected. 1 cc. of the formol treated lysate (after neutralization of the irritating formol by admixture of caustic soda) was injected subcutaneously in human subjects without any trouble to speak of. The author recommends this vaccine for a trial in practice.

W. J. Bais.

SASAKI (R.) & MANABE (E.). Ein Fall von Kinder-Dysenterie mit Darmblutung. **Dysentery in a Child with Intestinal Haemorrhage.**—*Oriental Jl. Dis. Infants.* 1926. Apr.–July. Vol. 1. No. 2–3. German Summary p. 80. [In Japanese.]

An account of a child five years and nine months old, with dysentery, passing much blood in stools, from which a Flexner bacillus was cultured. The blood serum was strongly Wassermann positive. The very bloody stools, in this case, possibly were due to syphilis, but the same authors observed another child, two years and five months old, with the same symptoms, in whom the blood serum was Wassermann negative.

H. M. H.

WELCH (T. B.). **Castor Oil *versus* Saline Aperient.** [Correspondence.]—*Lancet.* 1926. Sept. 25. p. 681.

Dr. Welch briefly recapitulates his successful results from the use of castor oil in bacillary dysentery. A full report appeared in *Kenya Med. Jl.* 1924. Aug., and has already received notice in this *Bulletin*, Vol. 22, 20.

H. M. H.

## MIXED AND UNCLASSED DYSENTERY.

WINKLER (Ludwig). *Lamblia intestinalis* und Cholecystitis. [*L. intestinalis* **and Cholecystitis.**]—*Med. Klin.* 1926. Aug. 27. Vol. 22. No. 35 (1133). pp. 1340–1341.

The author states that though hitherto *Lamblia intestinalis* has been denied pathogenicity, recent observations by various workers (a few of whom he refers to) have incriminated this protozoon as causative agent in catarrhal bowel complaints. It is certain, however, that the flagellate can also be found as a normal harmless inhabitant of the human gut. WESTPHAL and GEORGI had found it twice in 100 cases investigated by stomach tube. The author in 23 cases investigated by duodenal tube had found it once—in a case with anorexia and stomach derangement. The tube revealed a definite duodenitis with scanty lamblia. None was found in the stool. Besides entero-colitis, WESTPHAL and GEORGI had recorded cases indicating that lamblia could set up disease of the bile passages. The author now records a case (male aet. 26 years) of chronic relapsing entero-colitis with definite physical signs and symptoms of cholecystitis, from whom, by duodenal tube, was obtained thick turbid brownish yellow, bacteriologically sterile, gall bladder secretion, containing leucocytes, epithelial cells, mucous shreds and *Lamblia intestinalis*. These parasites were also found in the first clear yellow fluid withdrawn through the duodenal tube. At that time no lamblia were found in the stool which was free from blood and mucus. Later, during a period of diarrhoea, active and encysted lamblia were found in the stools. Culture of stool gave no pathogenic bacteria. Treatment by emetine, followed by a course of Carlsbad salts, was effective in ridding the patient of all symptoms. One month later the duodenal tube revealed no pathological changes. Under emetine the lamblia became scanty and motile forms were found only at rare intervals.

H. M. H.

PARODI (Silvio A.) & NIÑO (Flavio L.). La "Giardia intestinalis" en nuestro país. Su frecuencia y rol patógeno. [**The Prevalence and Pathogenic Action of** *Giardia intestinalis* **in Argentina.**]—*Semana Méd.* 1926. Sept. 16. Vol. 33. No. 37 (1705). pp. 715–718. With 1 text fig.

Infection by *Giardia intestinalis* appears to be comparatively frequent in Buenos Aires. Some statistics show a percentage of 10·1 in over 2,000 cases examined; others as much as 25 per cent. The prevalence is greater amongst children, and it is owing to disregard of age that the findings differ so widely. In adults, women are more attacked than men, because of their closer contact with the children.

Examination of 2,011 specimens of faeces at the Hospital de Clínicas revealed Giardia in 6·51 per cent. In 1·5 per cent. it was the only parasite found, in 2·63 in conjunction with *E. histolytica*, in 0·53 with *Trichomonas hominis*, and in 1·85 with Blastocystis.

The authors state that Giardia stools are of a fetid odour, diarrhoeic, yellow, with mucus and at times blood. The symptoms are colic, tenesmus and epigastric pain. The course is usually chronic with exacerbations. After trying various forms of treatment, stovarsol and Yatren 105 amongst them, without effect, he obtained cure of an obstinate case by the use of "Narsenol Poulenc."

Experimental administration to cats, rats, and other laboratory animals may result in death, and at autopsy the mucosa of the caecum, ascending colon, and the lower part of the small intestine is inflamed, ulcerated, and perhaps even necrotic, with petechial haemorrhages. The author is of opinion that Giardia has definitely a pathogenic action.

H. Harold Scott.

PAWAN (J. L.). **A Case of** *Giardia intestinalis* **treated with Stovarsol.**—*Ann. Trop. Med. & Parasit.* 1926. June 24. Vol. 20. No. 2. pp. 197–198.

Male, age 2½ years. Abdominal pain, fever, very frequent watery green stools, in which active *Giardia intestinalis* were found. Emetine subcutaneously ⅙ gr. on second and third days and ¼ gr. on fourth day was followed by only slight fall of temperature. There was no sign of formed stools. On fifth day 1 gr. stovarsol was given by mouth and emetine stopped. Twenty-four hours later, less frequent stools becoming formed, definite improvement in general condition and temperature normal. One grain stovarsol given in each of following three days, by end of which time motions faeculent and formed and no pains. Giardia still present in stools. Stovarsol given every other day for six days, at end of which time no Giardia found in (normal) stools. Stovarsol continued 1 gr. every third day for 14 days, at end of which time no Giardia found in stool, and still absent 28 days later.

The author notes DOBELL's statement that emetine has no effect whatsoever on *Giardia intestinalis* or any other "common intestinal flagellates," nor has methylene blue, as advocated by CASTELLANI. Salvarsan *per os* is said (BAYANT, CARR and CHANDLER) to have cured human cases and mice and rat cases (ROGERS, KOFOID, YAKIMOFF). But DOBELL finds cure in these cases inconclusive. [Apart from conclusive "cure," there is the obvious duty of clinical relief of symptoms, if possible, and as quickly as possible.]

H. M. H.

MALLIK (K. L. Basu). **A Case of Infection in an Infant with** *Giardia* (*Lamblia*) *intestinalis*.—*Indian Med. Gaz.* 1926. Sept. Vol. 61. No. 9. pp. 448–449. [1 ref.]

Hindu male baby, one year old, bottle fed from two months, delicate, and frequently subject to milk indigestion, was suddenly taken ill, with temperature 103·4° F., vomiting and diarrhoea. In spite of various remedies, condition worsened, until on the fifth day microscopical examination of mucus from stool revealed large numbers of Giardia, actively motile and encysted. Stovarsol 1 grain every four hours was given. There followed very rapid improvement and after a week's treatment no cysts were found in the stool.

H. M. H.

AGUILAR (Ricardo). **The Treatment of** *Balantidium coli.—Fourteenth Ann. Rep. Med. Dept. United Fruit Company. Boston, Mass.* 1925. pp. 246–248.

Pigs are the natural host of *Balantidium coli*, but it can thrive and produce lesions in the intestines of men and monkeys. The organism, first described by MALMSTEN in Stockholm in 1857, has been reported in the human being in many parts of both tropical and colder latitudes. Among 40 cases of balantidic infection in the Quirigua hospital, Guatemala, 34 were suffering from malaria, two from pneumonia, one from inguinal adenitis, one from furnuculosis, and two from severe uncinariasis. All but four of the 40 cases harboured one or all of the following helminths, uncinaria, trichocephalus, and ascaris. All suffered from diarrhoea—5 to 16 stools daily. The stools of 14 contained pus, or pus and blood, denoting ulceration of the bowel.

Balantidic infection is most prevalent at Quirigua in August, September and October, very few cases being reported in the other months. The rainy season—average rainfall 15 inches per month—is June, July, August and September. A feature common to all countries is that balantidic infection occurs among those closely associated with the handling of pigs or pork. Guatemala is no exception; there the pig is a common domestic animal.

The balantidium thrives in the liquid contents of the bowel; only encysted forms are found in the formed faeces.

The author believes that balantidium thrives best in people who are consuming excessive amounts of carbohydrates and insufficient quantities of protein foods and green vegetables.

*Treatment.* First—reduce the carbohydrate intake. Second—stovarsol is apparently "a specific." It should be given as a tablet (0·25 gramme) 2 or 3 times daily, after meals, for seven days. In all 40 cases, after five days' stovarsol treatment as above, the stools were negative for *Balantidium coli*. Many of these cases examined two months later were found to be still free of pus, blood, or *Balantidium coli*.

The author thinks it possible that chickens and birds may become hosts of *Balantidium coli*. He fed two canaries on lettuce contaminated with cyst-containing stools. Both developed diarrhoea—pus, blood, and *Balantidium coli* being found in the evacuations—and died within two days.

*Balantidium coli* is probably transmitted by means of faecal-contaminated water or green vegetables, as well as by mechanical means.

H. M. H.

HERMITTE (L. C. D.), SEN GUPTA (S. C.) & BISWAS (T. N.). **The Therapeutic Value of Stovarsol administered by Mouth in Parasitic Infections of the Bowel, with Special Reference to its Rapidly Curative Effect in Human Balantidiosis. (A Contribution from Assam.)**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1926. June 17. Vol. 20. No. 3. pp. 206–212. [19 refs.]

The authors have had good results from stovarsol in amoebic dysentery, lambliasis, and trichomonas infections, and in this interesting paper they report results of its use in four cases with mucosanguineous

stools containing *Balantidium coli*. The cases are fully described. Léon BERNARD and THOMAS (1924) reported cure of one case of balantidiasis by stovarsol.

Case 1. Emetine, E.B.I., N.A.B. (by mouth), had no effect whatever. Death occurred before stovarsol was tried. Rectal injection of quinine (reported as effective in balantidiasis) was not tried.

Case 2. Balantidium and trichomonas disappeared after the third dose of stovarsol (12 grains in all) and a further 70 days of repeated stool examinations yielded neither of these parasites, even after saline purges.

Case 3. Balantidium disappeared after fourth day of treatment with stovarsol (16 grains in all) and daily examination of stool for 62 days yielded constantly negative results.

Case 4. Balantidium was not found again after third dose of stovarsol (12 grains in all) and was absent during the 11 days following and preceding this patient's death from broncho-pneumonia. (Lamblia disappeared for good after the second dose of stovarsol (8 grains in all).

Hitherto the therapy of balantidiasis has been difficult, but from these results the authors, not unreasonably, claim that in stovarsol one has an excellent remedy for balantidial enteritis. It is a safe drug, [death from arsenical dermatitis following its use is known], easy to administer (by mouth). It is a good general tonic and has a marked influence on the haemopoietic system. They are sure that with a wider use of the microscope in Assam hospitals, balantidiasis will be found to be much more common among coolies than hitherto known or believed.

H. M. H.

DEVESA. [**Balantidium Dysentery in Spain.**]—*Arch. Esp. de Enf. del Aparato Digestivo.* Madrid. 1926. Apr. Vol. 9. p. 228. [Summarized in *Jl. Amer. Med. Assoc.* 1926. July 31. Vol. 87. No. 5. p. 367.]

Report of two cases, the first to be discovered in Spain. Both were cured by emetine and neoarsphenamine, aided by enemas of bismuth. Since MALMSTEN discovered the parasite in Stockholm in 1857, no infections in human beings have been recorded in England, and only five in France.

H. M. H.

WITHERSPOON (Jack). Report of a Case of Amebic Dysentery with Liver Abscess and Rupture into the Pleura.—*Southern Med. Jl.* 1926. Sept. Vol. 19. No. 9. pp. 673–675. With 3 text figs. [6 refs.]

## PLAGUE.

GAÏSKY (N.) & ALIFONOV (S.). Epidémie de peste pulmonaire à Baïkodam de la région sablonneuse de la steppe de Kirghiz. [**Epidemic of Pneumonic Plague in the Sandy Region of the Kirghiz Steppe.**]—*Rev. Microbiol. et Epidémiol.* 1926. Vol. 5. No. 3. French summary pp. 229–230. [In Russian pp. 133–136. 2 refs.]

The centre of this outbreak was 100 kilometres to the north of the Caspian Sea at Baikodam. The epidemic lasted a month, from the middle of February to the middle of March, 1926. There were 28 cases of pneumonic plague, all fatal. In the first case, a Kirghiz woman, there was present a submaxillary bubo. In all the others, the infection spread by contact between families. It was considered that the original infection came from mice, as there was an epizootic among mice and gerbilles in the Baikodam region.

J. H. Tull Walsh.

SILPENOV (I. V.). **The Plague Outbreak in Kzil-Kuga Rayon, the Uralsk Government, in the Winter of 1925.**—*Rev. Microbiol. et Epidémiol.* 1926. Vol. 5. No. 3. English summary pp. 232–233. [In Russian pp. 147–151.]

This was an epidemic of pneumonic plague, north of the Caspian Sea, without buboes, even in the first case. The actual source of infection was not traced, but it was supposed to have come from contact with rodents. The outbreak lasted from November 16th to December 27th, 1925. Forty persons were attacked; all died. The author considers that this high mortality was due to lack of local medical organization. Anti-plague detachments arrived, after some delay, about December 10th.

J. H. T. W.

i. KLODNITZKI (N.). Voprosi Epidemiologii Astrachanskoi Chumi. **Problems of Plague Epidemiology in Astrakhan.**]—*Guigiena & Epidemiologia.* Moscow. 1926. Vol. 5. No. 1. pp. 43–55. French summary p. 145. [11 refs.]

ii. GAISKI (N.). Chuma u Suslikov po Vremenam Goda. [**Plague in Susliks in relation to Season.**]—*Vjestnik Microbiologii & Epidemiologii.* 1926. Vol. 5. No. 1–2. pp. 3–19. French summary pp. 103–105. With 1 fig. & 6 diagrams. [14 refs.]

i & ii. While it is universally agreed that sisels (susliks) and mice (both domestic and wild) are directly responsible for the human plague outbreaks in South-east Russia, much controversy still exists as to the permanent reservoir of infection. As gathered from the discussions at the Saratov Anti-Plague Conferences, some observers have not emancipated themselves from the idea that the rodents might become periodically infected by gnawing buried corpses of human plague victims. More stress should be laid upon the contention of Klodnitzki that the small susliks (*Spermophilus musicus, mugosaricus*, etc.) are not the permanent carriers of plague but receive infection from the larger species. Klodnitzki (i) suspects now the *Cynomys fulvus* (black

*spermophilus* or small prairie dog) to be the fountain-head of infection. However, the systematic studies of NIKANOROFF (this *Bulletin*, Vol. 23, p. 184) and Gaiski (ii) leave little doubt that plague is perpetuated among the small susliks. Gaiski suggests, as a result of his experiments undertaken throughout a whole year, that the continuation of the disease among sisels without their dying out wholsesale is due, not as much to a gradually developing specific immunity, as to seasonal changes of susceptibility to infection.

Wu Lien Teh and R. P.

ii. From July 20th, 1924, to June 20th, 1925, the author experimentally infected 242 spermophiles with plague. Of these, 30 were inoculated during full hibernation. The conclusions drawn from the experiments are: (1) The susceptibility of spermophiles to plague varies according to the season, decreasing in winter, increasing in summer. In the bodies of hibernating animals *B. pestis* seems to lose virulence and is less easily cultivated; the type of plague may even be localized and atypical. Epizootics of plague generally begin after hibernation and the months of June and July are most dangerous on account of the large number of septicaemic forms.

J. H. T. W.

WAGNER (J.) & JOFF (I.). O Blochach Suslikov (i Tushkanchikov) v Sviazi s ich Roliu v Rasprostranenii Chumi v Privolshskich Stepiach. [**On the Fleas of Susliks (and Jerboas) and their Rôle in the Spread of Plague in the Volga Steppes.**] — *Vjestnik Microbiologii & Epidemiologii*. Saratov. 1926. Vol. 5. No. 1–2. pp. 57–100. German summary pp. 108–123. With 24 figs.

This exhaustive study contains a detailed description of the fleas found on the South-Russian sisels and jerboas. The following flea list may be given for three species known to suffer from natural plague :—

| *Citellus musicus* : | *Citellus mugosaricus* : | *Alactaga saliens* : |
|---|---|---|
| *Ceratophyllus tesquorum.* | *Cer. tesquorum.* | *Mesopsylla tuschkan.* |
| *Ctenophthalmus pollex.* | *Cten. pollex.* | *Ceratophyllus (Ophthalmopsylla) volgensis.* |
| *Neopsylla setosa.* | *Cten. breviatus.* | |
| | *Neopsylla setosa.* | |

Evidently the suslik fleas do not live constantly on their hosts, but dwell partly in the burrows; this holds apparently true of the species of *Neopsylla* and *Ctenophthalmus* which possess rudimentary eyes.

Investigations upon the flea rate at different seasons of the year are not complete, so far as the numbers found in the nests are concerned. The rate of fleas found on the rodents themselves seems to increase in the course of the summer.

The authors emphasize that only in rare cases the fleas of *Citellus musicus* were seen to pass over to the jerboas and *vice versa*; though both species live in close contact. [It is interesting to remember in this connexion that, according to NIKANOROFF,* no plague transition from one species of South-Russian rodents to another has yet been observed under natural conditions] [*v.* this *Bulletin*, Vol. 23, p. 618 (GOLOV & JOFF)].

Wu Lien Teh & R. P.

* *Vj. Microbiol. & Epidemiol.* 1925. No. 3. p. 34.

SKORODUMOFF (A.). O Tarabaganei Chume v Zabaikalie. [**Tarabagan Plague in Transbaikalia.**]—*Mikrobiologicheski Jl.* 1925. pp. 210–212. [2 refs.]

——. Obsledovanie Chumnogo Ochaga Zabaikalia Letom 1925 Goda. [**Observations on the Transbaikalian Plague Area in Summer 1925.**]—*Guigiena & Epidemiologia.* Moscow. 1926. Vol. 5. No. 2. pp. 47–50. French summary p. 108. [1 ref.]

——. Ob Organizatsii Protivochumnogo Diela v Sibiri. [**Organization of Anti-Plague Work in Siberia.**]—*Trans. of the 1925 Saratov Anti-Plague Conference. Saratov. 1926.* pp. 230–237. German summary p. 286.

——. K Voprosu o Desinfektsii Tarabaganich Shkurok v Zabaikalii. [**On the Disinfection of Tarabagan Skins in Transbaikalia.**]—*Ibid.* pp. 237–243. German summary p. 287. [7 refs.]

Skorodumoff's observations on disinfection of marmot skins may be noted. He found the disinfecting chamber at Borzia, where the permanganate-formalin method was adopted, insufficient; skins of plague-infected guineapigs were found to contain *B. pestis* after having been exposed in the chamber; plague cultures, bed-bugs, fleas, lice and ticks were alive after exposure. Good results were obtained in the steam-formalin chamber at Kiachta if a temperature of 60° C. was maintained for one hour. At a temperature of 52-56° C. plague cultures were not killed in an hour, and positive results were obtained from the exposed skins of infected guinea-pigs and mice. The quality of the furs was apparently not impaired by the disinfection.

Wu Lien Teh & R. P.

LEGER (Marcel). La peste: rôle de l'homme dans la conservation du virus. [**Plague: the Rôle of Man in the Conservation of the Virus.**]—*Paris Méd.* 1926. June 5. 16th Year. No. 23. pp. 552–556. [24 refs.]

This paper gives further examples of cases of plague in which the author regards man as the source of infection. From numerous observations he concludes that man is liable, on the same level as rodents, to carry on and perpetuate plague infection. He states as one proof that three natives of Dakar were carriers of *B. pestis* without showing any signs of illness. They were kept under observation during about one month. Freed from isolation to ordinary life, they continued to enjoy perfect health [*v.* this *Bulletin*, Vol. 21, p. 878 (LEGER)].

J. H. T. W.

WILLIAMS (C. L.). **Modern Trends of Plague Epidemiologic Research.**—*Milit. Surgeon.* 1926. Aug. Vol. 59. No. 2. pp. 155–164. [12 refs.]

A good general account of modern lines of research in plague. The author writing of "resolving plague" in rodents records that, in New Orleans, two rats showing signs of resolving plague were discovered, in which pestis bacteraemia was demonstrated. Further, by inoculating a large number of rats with plague, it was shown that a few of them, after recovery from the acute infection, continued to harbour plague bacilli in the blood for as long as 30 days after the inoculation (longer periods

were not tested). The proportion was small —four out of 890 rats, or ·45 per cent. This suggests important possibilities and some results will be published shortly in the *American Journal of Tropical Medicine.*

J. H. T. W.

BICKOV (S. G.). **On the Influence of Plague over the Sexual Sphere of Woman.**—*Rev. Microbiol. et Epidémiol.* 1926. Vol. 5. No. 3. English summary p. 236. With 3 figs. on 1 plate. [In Russian pp. 165–174. 15 refs.]

These notes on the pathology of plague as it affects the female sex organs come from the Clinic of Obstetrics, Saratov University. Pregnancy is stopped, and mother and foetus perish. There is haemorrhage into the serous layer of the uterus. Under the microscope the uterus shows hyperaemia and extravasations. In the blood vessels plague bacilli occur. During menstruation, clots of blood are found in the mucous membrane and the alterations in the muscular layers of the fallopian tubes are identical with those in the uterus. The plate shows sections of: (1) vascular layer of uterus; (2) submucous muscular layer of uterus; and (3) graafian follicles in the stage of degeneration.

J. H. T. W.

PASO (Juan Roberto). Los procesos neumónicos en la peste ganglionar. Estudio de la bronconeumonia pestosa secundaria (bronconeumonia bubónica). [**Secondary Broncho-pneumonia in Bubonic Plague.**]—*Semana Méd.* 1926. Aug. 26. Vol. 33. No. 34 (1702). pp. 505–525. With 9 text figs. & 2 charts.

Since 1900, when there was an epidemic of plague in Buenos Aires, numerous sporadic plague cases have occurred, showing that plague infection does not disappear with the ceasing of an epidemic. The author has opportunities for studying such cases in the Hospital for Infectious Diseases. He has investigated in particular the broncho-pneumonia occurring in cases of bubonic plague, its morbid anatomy and histology. Two fatal cases are described in great detail, and the text figures illustrate the condition of the lungs and the minute pathology disclosed by the microscope. The paper concludes with references to the work of other observers and a general review of secondary plague pneumonia.

J. H. T. W.

SHARP (N. A. Dyce). **A Note on the Treatment of Plague in Nigeria with "Bayer 205."**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1926. Feb. 18. Vol. 19. No. 8. pp. 482–484. With 9 charts.

In Lagos during the last four months of 1924 over 150 cases of plague were admitted into the Infectious Diseases Hospital. Many arrived in a moribund condition. The death-rate was about 80 per cent. Methods of treatment included: intravenous injections of anti-plague serum in doses of 50 to 250 cc.; injections of a solution of iodine (twice as much iodine as in tinct. iodi mitis) in doses of 10 to 60 minims; injections of N.A.B. and of perchloride of mercury $\frac{1}{8}$ gr. to $\frac{1}{4}$ gr.; and also carbolic acid in 1 minim doses and salol 5 gr., by the mouth. Towards the end of the epidemic, owing to unsatisfactory results, injections of "Bayer 205" were tried. Altogether 12 cases were thus

treated, and of these, three were in a hopeless condition when admitted. Of these 12, four died including the three moribund cases. The fourth case, an old woman of 80, failed to respond to any treatment. In the eight cases which recovered, fall of temperature and improvement in general conditions occurred. The author states that "Bayer 205" in small doses is free from danger. He thinks that repeated small doses are of more benefit than one large one.

J. H. T. W.

BHARADWAJ (A. C.). **The Treatment of Bubonic Plague with Intravenous Injections of Iodine.**—*Indian Med. Gaz.* 1926. Feb. Vol. 61. No. 2. pp. 63–64.

The author uses a solution of 18 grains of iodine with 36 grains of iodide of potassium in 4 ounces of normal saline, and injects, intravenously, 5 to 10 cc. in one dose daily for four days. He has treated 100 cases with about 80 per cent. cures. Early cases do well, but serious cases in an advanced stage do not respond to this treatment. With this treatment he always gives the following mixture, every four hours :—

| | |
|---|---|
| Pot. iod. ... ... ... ... | gr. v |
| Sp. Ammon. Aromat. ... ... | m. xx |
| Tinct. Strophanthi ... ... | m. ii |
| Sp. Vini Gallici ... ... ... | drs. ii |
| Aqua ad ... ... ... ... | oz. i |

[*cf.* this *Bulletin*, Vol. 22, p. 378 (MARSHALL and other references)].

J. H. T. W.

GRIMES (C.). Du traitement de la peste bubonique par les injections intraveineuses d'iode colloidal. [**Treatment of Bubonic Plague with Colloidal Iodine.** *Bull. Soc. Path. Exot.* 1926. June 9. Vol. 19. No. 6. pp. 407–409.

Seven cases of bubonic plague were treated with intravenous injections of a preparation of colloidal iodine known as "Neo-Riodine." The doses varied between 30·0 cc. and 60·0 cc. in 24 hours, 10 cc. every 4 or 6 hours, until a normal temperature appeared. Six of the patients recovered ; one died, and in this case there was some suppression of urine, with disease of the kidneys.

J. H. T. W.

TURKHUD (D. A.). **The Treatment of Plague ; a New Suggestion.**—*Indian Med. Gaz.* 1926. Aug. Vol. 61. No. 8. pp. 387–389. [5 refs.]

Having reviewed the treatment of plague with the anti-serum, tincture of iodine and plague "bacteriophage," the author introduces his new suggestion which is based on the following argument. The addition of 2 to 5 per cent. of sodium chloride to fresh bacterial cultures will produce "involution forms." Intravenous injections of salt solution, such as have proved harmless in man, should "not only help to relieve the congestion of the liver, spleen, and other internal organs, but, by flushing the kidneys and the intestines, also remove the toxins from the general circulation" and are "extremely likely" to have a specific action on the bacilli themselves.

The author suggests that this treatment might be carried out on the following lines: A stock solution, ready for use, of sodium chloride pure 150 grammes, distilled water 1,000 cc., sterilized either in an autoclave or by boiling; temperature of solution at time of injection 100° F. Of this solution, 50 to 100 cc. should be injected at a time with aseptic precautions. The injections might be given once or twice a day, according to the conditions of the case, strict watch being kept for the increase of bile pigments in the urine.

J. H. T. W.

INGRAM (A.) & PIRIE (J. H. Harvey). **Report on Bacteriological Research Work carried out in Connection with Plague during 1925.**—*S. African Med. Rec.* 1926. June 12. Vol. 24. No. 11. pp. 252–257.

Several aspects of the plague problem in South Africa have been investigated:—

1. *Comparative Susceptibility to Plague of Various Veld Animals.* Prior to this work, natural plague infection was recorded in:—

(1) Common (Lobengula) gerbille.
(2) Multimammate mouse.
(3) Striped mouse.
(4) Long-eared mouse.
(5) Ground squirrel
(6) Eastern Karroo rat.
(7) Spring hare.
(8) Yellow mongoose.

Every *rodent*, including the seven above mentioned, and several other species, has been found sensitive to infection, showing some difference in degree between different species; all the veld rodents appear more sensitive than the common black rat. "Our present knowledge" points to the following as of practical importance:—

(1). On the high veld.—The gerbille, multimammate mouse and the ground squirrel.

(2). On lower, more bushy country.—The striped mouse. The Karroo rats, important as regards number, and perhaps the most sensitive to experimental infection, appear to escape when an epizootic rages among gerbilles living close to them. The reason seems to be that the flea parasites differ from those of the gerbilles. For a similar reason the spring hare is not of much importance in spreading infection.

Two carnivores living in close relations with rodents, which they eat, have been observed dying in numbers in the wake of a plague epizootic. They are the suricat and the yellow mongoose. Experimentally, the suricat is easily infected with plague, by inoculation and by feeding; it has not been found possible to infect or kill a mongoose although enormous doses have been employed. This animal should be protected. The suricat probably plays little part in actually spreading the disease.

Nine strains of South African *B. pestis* (two from rodents, seven from human beings) have been compared with human strains and rat strains from Bombay and human strains from Ceylon (Japanese ship), Manchuria and Nairobi. All these strains had the microscopic and cultural characters of *B. pestis*. With some differences of virulence, all the strains gave the characteristic pathological picture of plague when suitable animals were infected either by inoculation or scarification. Agglutination tests were positive, but in varying degrees. Field

experiments showed that plague could be spread among gerbilles by laying down infected food, and by letting loose infected animals. Dr. Pirie also reports what he believes to be a new disease in gerbilles, due to a bacillus belonging to the diphtheroid group. The entomological work was done by Dr. INGRAM. Four fleas have been added to the list of those known to occur on veld rodents. Breeding, longevity and feeding experiments were carried out. In the transmission experiments the method employed was that of the Indian Plague Commission. The fleas tested were: *Dinopsyllus lypusus* (J. & R.), *Chiastopsylla rossi* (Waterston), and *Xenopsylla cridos*. The two first both conveyed plague from infected to uninfected rodents. The single experiment with *X. cridos* produced no result, the number of fleas (20) being probably too small.

Further work is contemplated. J. H. T. W.

JETTMAR (H. M.). **Notes on the Vitality of Plague Bacilli in Stained Smears.**—*Nat. Med. Jl. China.* 1926. Feb. Vol. 12. No. 1. pp. 1–8. [13 refs.]

The vitality of *B. pestis* has been studied by drying on splintered glass or cover slips in sterile Petri dishes, at room temperature. As soon as the smears began to dry at the border, they were passed through the flame 5 or 6 times until completely dry. The stains employed were those used in routine work. The vitality of the bacilli was tested on 28 young, full-grown guineapigs; average weight 400–600 gm. A little normal saline was poured on the slide and the film scraped off with the edge of another slide. Half the emulsion was injected subcutaneously into a guineapig, the other half was soaked up in a swab and rubbed into the shaved abdominal skin of another guineapig. The results of the experiments are contained in the following table:—

RECORD OF FOURTEEN MALE GUINEAPIGS INOCULATED ON OCTOBER 24TH, WITH THE EMULSION OF SMEARS FROM THE INGUINAL BUBO.

| Guineapig. No. | Wt. | Dried at room temperature. | Passed through Flame. | Fixed by Alcohol. | Staining. | Mode of infection. | Result. |
|---|---|---|---|---|---|---|---|
| 3 | 490 g. | 1 hr. | — | — | — | Subc. | Died after 7½ days. Typ. Bub. Pl. |
| 4 | 520 g. | 1 hr. | — | — | — | Intrac. | Remained well. |
| 5 | 400 g. | 10 min. | — | 30 sec. | — | Subc. | ,, ,, |
| 6 | 500 g. | 10 ,, | — | 30 ,, | — | Intrac. | ,, ,, |
| 7 | 400 g. | 15 ,, | 7 times | — | — | Subc. | Died after 6 days. Typ. Bub. Pl. |
| 8 | 420 g. | 15 ,, | 7 ,, | — | — | Intrac. | Died after 8½ days. Typ. Bub. Pl. |
| 9 | 610 g. | 20 ,, | 6 ,, | — | Loeffler's Meth. | Subc. | Remained well. |
| 10 | 470 g. | 20 ,, | 6 ,, | — | ,, | Intrac. | ,, ,, |
| *11 | 550 g. | 25 ,, | 7 ,, | 30 sec. | ,, | Subc. | — |
| 12 | 500 g. | 25 ,, | 7 ,, | 30 ,, | ,, | Intrac. | Remained well. |
| 13 | 520 g. | 30 ,, | 7 ,, | — | 1 : 10 Car. fuchs. | Subc. | Died after 8½ days. Typ. Bub. Pl. |
| 14 | 550 g. | 30 ,, | 7 ,, | — | ,, | Intrac. | Remained well. |
| 15 | 550 g. | 35 ,, | 7 ,, | 30 sec. | ,, | Subc. | Died after 5½ days. Typ. Bub. Pl. |
| 16 | 540 g. | 35 ,, | 7 ,, | 30 ,, | ,, | Intrac. | Remained well. |

* Succumbed to pneumonia on November 2nd; no plague bacilli were to be found in the organs.

The action of direct sunlight on a thin film of plague bacilli on glass is especially marked. Bubonic pus dried on cover slips for four hours did not contain any living bacilli (KITASATO). Pus from plague dried at 28°–30° C. contained living bacilli after two days ; all dead after four days (KITASATO). Other workers' results are quoted. The German Plague Commission reported (1899) that after two days plague bacilli could no longer be cultivated from material dried on splintered glass.

J. H. T. W.

MARUYAMA (Y.). Ueber die Wirkung des Pestkoktantigens. [**Boiled Plague Bacilli as Antigen.**]—*Taiwan Igakkai Zasshi* (*Jl. Med. Assoc. Formosa*). 1926. May. No. 254. German summary p. 1. [In Japanese.]

The antigenic power of bacteria may be increased by heating to boiling point for a definite time. On the strength of this finding, the author prepared a vaccine of plague bacilli so heated, and compared its action with that of a vaccine prepared in the ordinary way. The effects were different in rabbits and guineapigs, which showed how dangerous it may be to transfer, without further trial, results obtained in one animal to another, and especially to the case of man.

W. F. Harvey.

ZANZUCCHI (A.) & VERCELLANA (G.). A proposito della reazione delle termoprecipitine negli organi pestosi umani conservati. [**Results of Thermoprecipitin Test in Preserved Organs from Human Plague Cases.**]—*Pathologica.* 1926. Apr. 15. Vol. 18. No. 414. pp. 185–187.

Extracts of the liver preserved in formalin gave positive results, showing the presence of plague precipitinogen. Extracts of kidneys and intestine preserved in alcohol also gave positive results. But the same reaction was obtained with other organs (preserved in formalin 5 per cent. ; alcohol 95°) which certainly had no connexion with plague.

J. H. T. W.

GRAHAM (J. D.). Enquête sur la peste pulmonaire dans l'Inde Britannique. [**Pulmonary Plague in British India.**]—*Bull. Office Internat. d'Hyg. Publique.* 1926. July. Vol. 18. No. 7. pp. 782–786.

This inquiry into pulmonary plague in India is an interesting report submitted to the "Comité de l'Office International d'Hygiène" at the October session, 1925. It contains nothing new.

J. H. T. W.

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ADOLPHI (Hans). Ueber die Pestepidemie in der Mandschurei im Jahre 1910.—*Arch. f. Hyg.* 1926. Vol. 97. No. 1–2. pp. 1–11.

——. Das Steppenmurmeltier und die Pest.—*Deut. Med. Woch.* 1926. May 14. Vol. 52. No. 20. pp. 829–830.

LUSTIG (A.). La peste bubbonica, i ratti e le pulci.—*Policlinico.* Sez. Prat. 1926. May 10. Vol. 33. No. 19. pp. 649–650. [2 refs.]

MACARTHUR (W. P.). Generalized Eruption in Plague. [Correspondence.]—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1926. Feb. 18. Vol. 19. No. 8. pp. 498–499.

PONS (R.). Procédé de diagnostic de la peste, post-mortem chez l'homme et chez les animaux.—*Bull. Soc. Path. Exot.* 1926. June 9. Vol. 19. No. 6. pp. 405–407. [5 refs.]

RAYNAL (M. J.). Epidémie de peste bubonique de Diégo-Suarez (juin-octobre 1924): dépistage *post-mortem*; sérothérapie et vaccination.—*Bull. Soc. Path. Exot.* 1926. July 7. Vol. 19. No. 7. pp. 592–604. [9 refs.]

SUWOROW (S. W.). Desinfektion- und Desinfektionversuche mit Chlor von Wohnraumen bei der Pest und Desinfektion der Sachen mit Schwefelkohlenstoff.—*Rev. Microbiol. et Epidémiol.* 1926. Vol. 5. No. 3. German summary p. 232. [In Russian pp. 143–145.]

TOURNIER (E.). La peste dans la province de l'Itasy.—*Bull. Soc. Path. Exot.* 1926. June 9. Vol. 19. No. 6. pp. 410–415.

## CHOLERA.

ROGERS (Leonard). **The Conditions influencing the Incidence and Spread of Cholera in India.**—*Proc. Roy. Soc. Med.* (Section Epidemiol. & State Med.) 1926. July. Vol. 19. No. 9. pp. 59–91. With 14 text figs.

This is an interesting essay written with the author's usual care. The numerous charts and tables are of considerable value.

*Conclusions.*—" (1) In the 1817–19 and subsequent cholera epidemics in India, up to those from 1859 to 1871 described by Bryden and Cornish, the disease appears to have spread from its home in Lower Bengal over North-Western, Central and Southern India in a series of waves of two to four years' duration at somewhat irregular intervals, the endemic area, according to Bryden, being limited to Bengal and West Assam.

" (2) Since 1877 the monthly cholera mortality for every district in India has been recorded, furnishing far more detailed information than the army and gaol figures of Bryden's time; but they have not hitherto been utilized for a comprehensive study of the incidence and spread of cholera in India, such as is attempted in this paper.

" (3) A study of the average monthly cholera incidence and rainfall, temperature and humidity in a diagram and four maps of the seasonal incidence in forty-five divisions of India, shows: (*a*) No uniform relationship with rainfall, as the disease during the south-west monsoon is at its maximum in most parts of India, but at its minimum in Lower Bengal; but it shows (*b*) a regular great decline or disappearance of the disease in all parts of India when the absolute humidity falls to or below 0·400, such great dryness of the atmosphere preventing the epidemic prevalence of the disease. The months in which cholera first shows a great increase after the winter quiescent period in North-West and Central India are those in which the absolute humidity first rises to over 0·400, the seasonal increase in most parts of India being thus explained quite irrespectively of any spread from Bengal.

" (4) The average annual incidence of cholera is highest in Assam, Lower Bengal, Bihar and the eastern sub-Himalayan divisions of the United Provinces of Northern India, and in South-east Madras; all areas with few or no months in absolute humidity below 0·400, and consequent continued prevalence of the disease throughout the year.

" (5) The present endemic areas, as shown by the disease never having been absent for a single year in three recent decades, include the areas of high incidence, just mentioned, of Bengal, the United Provinces and Madras, together with the low-lying west coast of Bombay, with the constant absolute humidity of over 0·400, so the endemic areas are now far more extensive and scattered than the parts of Assam and Bengal indicated by Bryden as late as 1869.

" (6) The epidemic areas, in which severe outbreaks occur frequently after a year or two of complete absence of the disease, include the south and west of the United Provinces, all the Punjab, the Sind, Gujerat and Deccan divisions of Bombay, and the whole of the Central Provinces. The spread of epidemics in these areas has been studied by means of curves of the annual incidence in forty-five divisions for forty-five years, and in as many yearly maps of the distribution and months of the first annual increase, maximum prevalence and decline for each year.

" (7) A map showing the number of epidemic rises in each area, and those in which a rise occurred in the same years in contiguous areas, shows a larger number of epidemics in the United Provinces than in Lower Bengal, so they could not all have originated from Bryden's Bengal endemic area. The studies of the forty-five years' maps clearly show that a number of the increases originated in the endemic area of the United Provinces above

described, from which they spread over the Punjab with decreasing intensity in proportion to the distance of the divisions from the United Provinces, and the dryness of their climate. Similarly, it is shown that the Central Provinces in recent decades were sometimes invaded from the east, from the southern Orissa divisions of Bengal, occasionally from the north, from the United Provinces, and frequently also from the west from the Deccan divisions, contrary to Bryden's conclusion that cholera always spreads from Bengal to the north-west over the United Provinces, or to the south-west over the Central Provinces to Bombay, with the monsoon winds. The facts on which he based his theory are now explained by the effect of low absolute humidity in inhibiting epidemics described in this paper.

"(8) The three most severe epidemics of modern times are considered in the light of the foregoing data, and in connexion with the meteorological conditions associated with them. The diffusion of the 1875–77 epidemic is mapped out and shown to have spread, largely through pilgrims, from separate endemic foci, north-west from Bengal and the United Provinces, east and south from Bombay, and north from Ceylon or Southern Madras. In each area of very high incidence the epidemic was associated with great deficiency of the previous rainfall, accompanied by drought, by bad water supplies, and often by famine. The epidemic of 1891–92 was spread mainly in the first year by a rarely occurring great Ganges pilgrimage, aided by deficient rains, and in the second year by continued deficient rainfall, and especially by the Hardwar pilgrims. The most severe epidemic of all, in 1900, was once more associated with very exceptional failure of both the monsoon and the succeeding winter rains over very large areas of India. A table is also given of the epidemic prevalence in every other of the forty-five years in which the total cholera mortality in India was much over the average. Out of twenty-five affected areas, in no less than twenty-four previous greater or less deficiency of the rains preceded the cholera exacerbations, and in the remaining area in the United Provinces in 1894 very exceptionally high humidity throughout the winter months was followed by a unique early recrudescence of the disease culminating in an epidemic.

"(9) By watching the climatic conditions influencing the seasonal and annual incidence of cholera in any area to which attention is now drawn, increased or epidemic prevalence should usually be foreseen in time to enable steps to be taken to lessen its spread by pilgrims and other travellers, by means of inoculating them against the disease before attending religious and other gatherings in cholera-infected districts. The Punjab, Sind, Gujerat and Deccan divisions of Bombay and the Central Provinces, so liable to invasion by epidemics, have largely in their own hands this simple means of lessening their cholera mortality. The sanitation, and especially the provision of a pure water supply, in all important pilgrim centres, should be a first charge on imperial and provincial revenues under reliable sanitary administration."

J. H. Tull Walsh.

REPPER (E. N. D.). **An Epidemic of Cholera amongst Ballast Train Coolies in Bengal.**—*Jl. Trop. Med. & Hyg.* 1926. July 1. Vol. 29. No. 13. pp. 196–197.

The author describes a ballast train, consisting of 35 to 40 open trucks. The train moves along the lines carrying ballast, bricks, sleepers, etc. The workers, 160 to 180 coolies—male, female and children—live on the train; many are born on the trains and live there all their lives. In March cholera attacked three ballast trains carrying, in all, over 500 coolies. The trains were taken to a suitable place for a cholera camp and segregated to prevent absconding and spread of the

infection by the frightened coolies. Among 509 coolies there were 40 cases and 17 deaths, considered a satisfactory result when the great difficulties the staff had to contend with are taken into account. The infected refused to go into the cholera huts and were treated in the wagons. They gave trouble over taking medicine and intravenous injection of saline solution had to be abandoned as the patients and their friends created so much disturbance. Subcutaneous injection was managed in the majority of cases; but only by holding the patient down and getting the solution in as quickly as possible. The epidemic subsided 48 hours after the trains were segregated in the camp.

J. H. T. W.

TOMB (J. Walker). **The Treatment of Cholera.**—*Lancet.* 1926. Oct. 2. pp. 721–722.

This letter contains a summary of various well-known methods of treating cholera in each of the stages of the disease. The author also alludes to the value of anti-cholera vaccination which conveys immunity lasting for several months. He concludes with a renewed recommendation of the "essential oils mixture"—*v.* this *Bulletin*, Vol. 23, p. 613 (YUI).

J. H. T. W.

PERUZZI (Mario). Fenomeni fermentativi e caratteri morfologici nella diagnosi dei vibrioni colerici e colerasimili. Ricerche sperimentali con una tavola di microfotografie. [**Some Fermentative and Morphological Characters of Cholera and Cholera-like Vibrios.**] —*Ann. di Med. Nav. e Colon.* 1926. July–Aug. Year 32. Vol. 2. No. 1–2. pp. 1–16. With 9 figs. on 1 plate. [15 refs.]

Section I describes methods recently described by CASTELLANI and others, of culture of organisms in symbiosis or close association. The author applies these to diagnostic differentiation. The types of association illustrated are :—

(1). Of one organism, producing only acid in a carbohydrate medium with one producing no acid, and giving both acid and gas.

(2). Of one organism producing both acid and gas in a carbohydrate medium, with one already grown some days, but not producing either, and giving a partial fermentation of acid without gas.

(3). Of one organism producing no gas in a carbohydrate medium, with another also producing no gas, and giving a fermentation with gas.

Differentiation of cholera-like organisms from cholera organisms by method (1) is usefully supplemented by method (3), a fact which is brought out in tables II and III.

Section II deals mainly with a new enrichment medium for the isolation of vibrios and for their morphological study. The technique of preparation and use is as follows :—

(1) Add to nutrient bouillon 10 per cent. sugar-free neutral gold label gelatin; (2) Neutralize accurately; (3) Heat together in a water bath, and boil for 10 min.: starch 1 gm.; asparagin 1 gm.; sod. chloride 1·5 gm.; water 100 cc., with frequent shaking to obtain a homogeneous suspension, (4) Add 10 cc. of the gelatin, 3 cc. 5 per cent. sod. carbonate and freshly prepared neutral solution of purified litmus to give the requisite colour; (5) Distribute 10 cc. quantities in test tubes; (6) Sterilize at 110° C.;

(7) Sow one loop test faecal suspension in 10 cc. medium; (8) Shake; (9) Incubate in the inclined position; (10) Examine microscopically after 7–8 hours incubation, or earlier; (11) Examine for acidification and pellicle formation; (12) Stain with carbol fuchsin or Giemsa and examine.

Section III deals with the polymorphism and structure of vibrios. The author concludes that cultural forms of cholera and cholera-like vibrios show: (1) differentiated chromatic bodies, presenting during division not only the appearances, but the behaviour of a nuclear apparatus; (2) the presence of sporiform bodies capable of development in linear form and branched symmetrically tripod forms with characters which suggest the affinity of vibrios to the *Fungi imperfecti* and particularly to the genus *Vibriothrix*.

W. F. Harvey.

BALFEANU (I.). **The Receptor Structure of** *Vibrio cholerae* (*V. comma*) **with Observations on Variations in Cholera and Cholera-like Organisms.**—*J. Path. & Bact.* 1926. July. Vol. 29. No. 3. pp. 251–277. With 6 figs. on 1 plate. [34 refs.]

The theory here investigated is given as follows: (1) The motile bacillus possesses two distinct kinds of agglutinable substances, one labile, the other stable when subjected to 100° C., or to dilute acids, or absolute alcohol; (2) The labile factor and its agglutinins are responsible for agglutination in large loose flocculi; the stable factor and its agglutinins are responsible for agglutination in small compact granules; (3) The isolated flagella react as if composed entirely of labile material; (4) The serum agglutinins for the labile antigen react in an inverse way and resist a temperature of 70° C. for 20 min.; those for the stable antigen are inactive after such treatment; (5) The non-motile races of normally flagellate organisms contain only stable antigen.

The stable agglutinins for the body substance of the organism are denominated "somatic" ("O") agglutinins and those for the flagella, labile "H" agglutinins.

PART I. *Summary.*—(1) *V. cholerae*, like *B. proteus* and bacteria of the typhoid-paratyphoid group, contains stable "somatic" ("O"), and labile "flagellar" ("H") antigens.

(2) The type of agglutination of these antigens is compact-granular and flocculent respectively, although the contrast between them is not so sharp in *V. cholerae*, which is only monoflagellate, as it is in the proteus and typhoid-paratyphoid groups. If, however, the cholera organism suspension be composed of specially collected flagella, it agglutinates then in characteristic flocculi. Such a flagellar suspension induces in the rabbit the production of a serum which reacts almost exclusively with the flagellar labile constituents of an ordinary suspension of vibrios.

PART II. Is concerned with a search for variants among the vibrios, with special reference to changes in the receptor factors here investigated. The strains used (7 *V. cholerae*, 2 El Tor, 1 *V. paracholerae*) were examined for their general cultural, biochemical and serological properties.

*Summary.*—Three kinds of colony variants were obtained which were readily distinguishable from the smooth round uniform transparent normal colonies. Of these variants I and II reverted with subculture on agar and variant III (opaque) reverted, though slowly, to normal. The opaque variant III formed a tough pellicle on liquid media, was non-motile, stained rather badly, appeared normal except for the absence of flagellum, and was surrounded with a mucoid material

which did not form a true capsule. It emulsified only with great difficulty, and that in distilled water; it became slightly unstable with the addition of salt to 0·85 per cent. No heat-labile flagellar flocculating "H" agglutinable substance could be demonstrated in suspension, but the heat-stable somatic granulating "O" factor was well represented. Serum produced with this opaque variant, however, contained "H" or flagellar agglutinins, which showed that some "H" antigen remained. It made as efficient a receptor as the normal in complement fixation tests and appeared to have normal virulence for guineapigs. The action of anticholera serum for this opaque form, however, was less bactericidal than normal, and the acid agglutination optimum was found to be at a higher hydrogen-ion concentration.

W. F. H.

HIRSCH (Julius). Die anaerobe Züchtung des *Vibrio cholerae.* [**Anaerobic Culture of the Cholera Vibrio.**]—*Klin. Woch.* 1926. June 11. Vol. 5. No. 24. p. 1089. [3 refs.]

The cholera vibrio is generally classed as an obligate aerobe, and this is the case for ordinary methods of growth. In a protein-free medium, with asparagin as the only source of both carbon and nitrogen, chemical analysis of the metabolic products of cultivation show that the utilization of the asparagin is an *oxidation* process. But if glucose be added to such a medium, the utilization of the sugar is not an oxidation, but a *splitting* or *anoxybiotic* process. The formula for such a medium, which will give an equal amount of transformation of the sugar under both aerobic and anaerobic conditions, is: sod. chloride 0·5; pot. chloride 0·1; m/15 phosphate buffer solution (pH7·8) 20; calc. chloride 0·5; sod. asparaginate 0·89; glucose 0·9; water to 100.

It is easy to understand now, how the cholera vibrio can develop in the infected intestine under what are practically anaerobic conditions.

W. F. H.

HIRSCH (Julius). Zur Biochemie pathogener Erreger. Wachstum und Stoffwechselleistungen des *Vibrio cholerae* auf einfachen—chemisch definierten-Nährböden. [**Growth and Metabolic Products of** *V. cholerae* **in Simple, Chemically-defined Media.**]—*Ztschr. f. Hyg. u. Infektionskr.* 1926. Sept. 11. Vol. 106. No. 3. pp. 433–467. With 1 text fig. & 7 charts. [Numerous refs.]

The author deals in this paper with the chemical decomposition effected by the *V. cholerae* when supplied with chemically-defined compounds as the sole source of supply of its necessary nitrogen and carbon food requirements. His media were exclusively fluid, containing pure mineral salts and of definite H-ion concentration, to which the organic compounds were added. He finds:—

1. *V. cholerae* can utilize l-aspartic acid as the sole nitrogen and carbon source of all its metabolic and energy requirements.

2. The decomposition of the l-aspartic acid by *V. cholerae* is of the nature of an oxidation process, having ammonia, acetic acid and carbonic acid as end products.

3. On a glucose-asparaginate medium *V. cholerae* forms ethyl alcohol in small quantity, formic, acetic and lactic acids.

W. F. H.

POPESCO (Cornelia). Action préventive exercée vis-à-vis de la péritonite vibrionienne expérimentale par les plaquettes sanguines de lapins immunisés contre le vibrion cholérique. [**Prevention of Cholera Peritonitis by Blood Platelets of Immunized Rabbits.**]—*C. R. Soc. Biol.* 1926. July 9. Vol. 95. No. 24. pp. 420–421.

Blood platelets of rabbits immunized against *V. cholerae*, after thorough washing, are capable *in vitro* of powerfully agglutinating and lysing the vibrios to granules in presence of a little fresh normal serum. The blood platelets of normal rabbits under these conditions bring about agglutination but no lysis. The author now investigates their action *in vivo* :—

(1) Guineapigs inoculated intraperitoneally with two minimum lethal doses of vibrios mixed with blood platelets of an immunized rabbit all survive; with three doses they succumb in the proportion of 50 per cent.

(2) Guineapigs inoculated with two minimum lethal doses, mixed blood platelets of the normal rabbit, succumb in the proportion of 64 per cent.; with three doses they all succumb.

(3) Guineapigs inoculated at the same time as the above with a single minimum lethal dose of vibrios and no blood platelets all succumb.

W. F. H.

SUZUKI (Kiyoshi). Infectionsversuche mit Vibrio Kadikjöji. [**Infection Experiments with** *Vibrio kadikjö.*]—*Arch. f. Hyg.* 1926. Vol. 97. No. 3–4. pp. 141–161.

This paper deals at considerable length with the mode of infection in animals by *V. kadikjö*, a member of the much discussed El Tor vibrio group.

Some of the points which the author brings out are :—

1. The extraordinarily rapid passage of vibrios into the blood after intraperitoneal inoculation of guineapigs: this is a matter of only a few minutes.
2. The comparative ease with which infection is brought about in guineapigs after subcutaneous inoculation, with appearance of vibrios in blood, peritoneum, liver, spleen, bone marrow, kidney and large intestine, although not in stomach, small intestine or urine.
3. Inability to produce infection of guineapigs by feeding.
4. The high degree of toxic action shown in rabbits, but also in guineapigs.

The subject is largely treated from the point of view of the inclusion of this vibrio among the semiparasite organisms. "Semiparasite" organisms are characterized by their ability to give rise to successful infection with intraperitoneal, and their inability with other modes of injection. Their direct introduction into the blood stream can bring about toxaemia, but not commonly a septicaemia.

Subcutaneous inoculation is without result or at most is followed by a localized abscess. *V. kadikjö*, if it is to be called a semiparasite, differs then markedly from the majority in the ease with which infection is produced in the guineapig by subcutaneous and intracardiac injection. It may perhaps be considered then to stand especially high in the list of semiparasites.

W. F. H.

VERCELLANA (G.). La differenziazione del v. del colera dai colerasimili mediante un saggio di agglutinazione aspecifica. **Non-specific Agglutination of Cholera and Cholera-like Organisms.** —*Patologica.* 1926. July 15. Vol. 18. No. 417. pp. 418–419.

The author with ZANZUCCHI had succeeded, by a non-specific acid agglutination test, in differentiating the closely allied organisms *B. melitensis* and *B. abortus* (Bang.). Could the test also serve to differentiate cholera from cholera-like vibrios? His results were: With lactic acid the cholera-like strains always agglutinated in large stable flocculi and very rapidly. Cholera vibrios, on the other hand, either do not agglutinate at all, as was the case for Plava and El Tor strains, or agglutinate slowly in small unstable flocculi and in lower dilution than the cholera-like strains.

W. F. H.

TOMB (J. W.). **The Prevention and Treatment of Cholera by Essential Oils.**—*Jl. Trop. Med. & Hyg.* 1926. July 15. Vol. 29. No. 14. pp. 210–212.

A further record of the treatment of cholera with the mixture of essential oils—see this *Bulletin*, Vol. 23, p. 613 (YUI) and reference to Tomb.

J. H. T. W.

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ASCIONE (Guglielmo). Sull'azione patogena di un vibrione colerasimile nella cavia.—*Ann. d'Igiene.* 1926. Jan. Vol. 36. No. 1. pp. 1–23. With 5 text figs. [19 refs.]

HAHN (Martin) & HIRSCH (Julius). Gewinnung von Choleragift.—*Klin. Woch.* 1926. Aug. 20. Vol. 5. No. 34. p. 1569.

LEAGUE OF NATIONS. Health Organisation. A Memorandum on the Epidemiology of Cholera. [RUSSELL (A. J. H.).]—46 pp. [108 refs.] C.H. 339. 1925. July 31. Geneva. [*v.* this *Bulletin*, Vol. 22, p. 765.]

## ENTERIC FEVERS.

FREW (A. & BUTT (H. T. H.). **Symposium on Enteric Fever. Manifestations Peculiar to Natives.** *Med. Jl. S. Africa.* 1926. June. Vol. 21. No. 11. pp. 309–311.

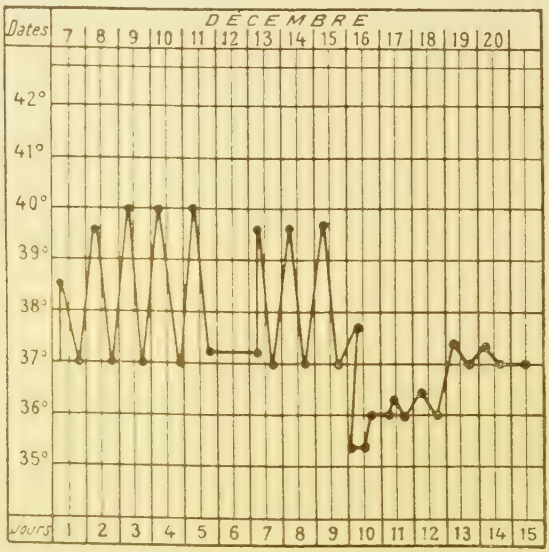
The title of this paper is somewhat misleading, as careful reading has not disclosed any "manifestation" that does not occur among other races and in other countries. The African native dislikes going into a hospital, so that frequently patients in an advanced and severe state are met with. One case is recorded of a native standing amongst a group of discharged boys awaiting his turn to be passed out to go home. One of the authors saw that the man had peritonitis and perforation. He was taken into hospital and died within 12 hours. Relapses are more common among natives than among whites from the difficulty in making a hungry native understand the dangers of dietary excesses, in which he is encouraged by well-meaning brothers with tins of sardines, etc.; partly, also, from the difficulty of keeping a convalescent native strictly in bed.

J. H. Tull Walsh.

i. UBAGO (M.). Typhoide du type inverse. [**Typhoid of Inverse Type.**] —*Rev. Méd. et Hyg. Trop.* 1926. Mar.–Apr. Vol. 18. No. 2. pp. 46–47. With 1 chart in text.

ii. ——. Para-typhoide "B." [**Paratyphoid "B".**]—*Ibid.* pp. 47–48. With 1 chart in text.

i. The author describes from Nicaragua a case of the "inverse type" of typhoid fever in which the temperature was high in the morning,

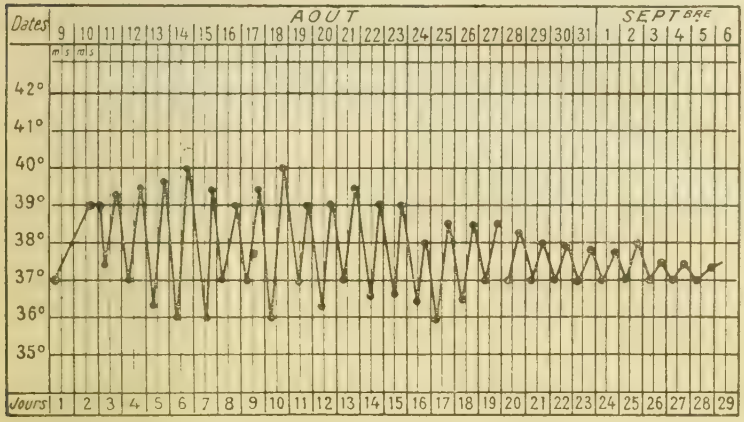



Temperature Chart of UBAGO's case of the "inverse type" of Typhoid Fever.

dropping to 37° C. in the evening for nine days and becoming sub-normal during the remaining days. The remarkable chart is given below.

The patient, a man aged 40, was at first treated as a case of malaria; but as quinine produced no effect, and in view of certain symptoms—headache, insomnia, foul tongue and stupor—the author considered whether it might not be a case of typhoid fever, and obtained a Widal reaction on the 9th day. On the 10th there occurred a marked haemorrhage from the intestines, with a fall of temperature to 35·5° C. The illness lasted 14 days and the man recovered.

ii. The case was that of a woman, aged 30, attacked with "fever" with high evening temperature, falling to normal or sub-normal, with




Temperature Chart of UBAGO's case of Paratyphoid B.

shivering which seemed to imply malaria. She also suffered from headache, insomnia and irregular pulse. No plasmodia could be found in the blood and quinine made no impression. A haemoculture showed the presence of *B. paratyphosus* B. Treatment—iodized vaccine of Rendue and Senez, lacteol and adrenalin. The illness lasted 29 days and the patient recovered.

J. H. T. W.

MÜNNICH (G.). Die chirurgischen Komplikationen des Typhus abdominalis. [**Surgical Complications of Enteric Fever.**]—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.* 1926. Vol. 30. No. 1. pp. 208–217.

A paper read in October, 1925, before the German Society for Tropical Medicine, in Hamburg. These complications are noted in most text-books and need not be noticed in detail. Actual cases seen by the author are described. Out of nine operations for intestinal perforation there were four recoveries.

J. H. T. W.

SISON (Agerico B. M.) & IGNACIO (Modesta). **Coleman Diet in Typhoid Fever.**—*Jl. Philippine Islands Med. Assoc.* 1926. Mar. Vol. 6. No. 3. pp. 84–94. [5 refs.]

The authors discuss the Coleman high caloric diet for enteric fever cases. A table of the maximum and minimum of calories required according to symptoms for such a diet is given, also tables of the small meals given every two hours. These tables take up much space; but an idea may be given by quoting three forms of diet as given :—

| | | | |
|---|---|---|---|
| 1. | Milk | 100 cc. | 70 cal. |
| | Cream | 10 cc. | 20 ,, |
| | Lactose | 15 gm. | 60 ,, |
| 2. | Milk | 100 cc. | 70 cal. |
| | Sugar | 5 gm. | 20 ,, |
| 3. | Milk | 100 cc. | 70 cal. |
| | Cream | 10 cc. | 20 ,, |
| | Lactose | 15 gm. | 60 ,, |
| | Egg soft-boiled | 1 gm. | 75 ,, |

"The effect of the high caloric diet was tried in so few cases by us that well-grounded conclusions cannot be drawn." The authors, however, state their experiments prove that the feeding of a typhoid patient with high caloric diet does not produce any deleterious effect. Indeed they believe that the diet has a highly favourable influence on the course of the disease.

J. H. T. W.

DOMINGO (P.) & VIDAL (J.). La vacunación antitifica "per os." [**Anti-typhoid Vaccination by the Mouth.**—*Rev. Méd. de Barcelona.* 1926. May. Year 3. Vol. 5. No. 29. pp. 465–468.

The first part of this paper gives a résumé of BESREDKA's experiments and the results of administration of dysentery bacilli by mouth, with and without the previous use of bile, and of his finding that the immunity effected is a local tissue-immunity in the intestine.

The authors applied a modification of these results to the use of *B. coli*, by the following technique: Twenty-four hour cultures on agar were emulsified in as small a quantity of water as possible. This was treated with 10 times its volume of alcohol and ether, equal parts. Setting aside for 12 hours sufficed for sedimentation of the bacteria. The supernatant fluid was decanted off and the bacillary deposit subjected to the action of sterile neutral ox-bile. After 12 hours at 5° C. the emulsion was centrifuged, and the bacteria diluted with a mixture in equal parts of water, 90 per cent. alcohol, and glycerin, 1 cc. to each centigramme of the bacteria. Six guineapigs received each 1 cc. of the vaccine so prepared, four of the same weight being kept as controls. Five days after 20 m.l.d. of a virulent *B. coli* were injected intraperitoneally. All the controls died in 24 hours, whereas all those vaccinated survived.

A vaccine of Eberth's bacillus was then prepared in the same way as the above, and six guineapigs were given three doses of 1 cc., four guineapigs being used for control. Ten days after the last dose, 1 [supposed] m.l.d. was injected intraperitoneally. In 48 hours the group of vaccinated animals were perfectly well with normal stools and temperature. The controls exhibited a rise of temperature with diarrhoea, but had completely recovered in 15 days. The experiment was repeated, but with 20 times the former infecting dose. All the

controls died within 48 hours, the others presented no ill-effects. These results are in course of application to man and will be the subject of a future report.

H. Harold Scott.

CORNWALL (J. W.). **On the Variation in Amount of Soluble Protein contained in Old Broth Cultures of** *B. typhosus.*—*Indian Jl. Med. Res.* 1926. Jan. Vol. 13. No. 3. pp. 483–490. With 3 charts in text.

This is an investigation into the variation in the quantity of soluble proteins and of compounds capable of being precipitated by lead acetate from culture, with lengthening incubation. The maximum is reached after 7 to 12 weeks and then falls. With diminution of the concentration of nutriment the generation time lengthens and reproduction is slowed down. The conclusions are drawn that: "The rise in the soluble protein curve indicates that a larger amount of protein derived from solution of bacterial bodies is being added to the fluid than is being abstracted by degradation of protein. The fall in the soluble protein curve indicates that degradation of protein by hydrolysis is proceeding more rapidly than protein is being added by solution of bacterial bodies."

W. F. Harvey.

CORNWALL (J. W.) & BEER (W. A.). **On the Persistence of** *B. typhosus* **in Rabbits after Intravenous Injection.**—*Indian Jl. Med. Res.* 1926. Jan. Vol. 13. No. 3. pp. 491–492.

Most rabbits which survive the injection free themselves from the bacilli entirely, but in some they may persist for several years, although the animal maintains good health. The chronic infection, however, may be contributory to the ultimate death of the rabbit. No indication of that infection is afforded by the agglutination titre of the serum. The history of three rabbits which were the subjects of chronic infection is given. Nos. 1 and 2 died of salmonella and bronchopneumonia infections respectively, and No. 3 was killed while still healthy. In each case the typhoid bacillus was recovered from the gall bladder and from the upper intestine or liver. These rabbits, as chronic carriers, appeared to differ from man, in that attempts to recover typhoid bacilli from the faeces were unsuccessful.

W. F. H.

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CALAZANS (Sebastião). Considerações sobre a febre typhoide em S. Paulo.—*Bol. Soc. Med. e Cirurg. de S. Paulo.* Brazil. 1925. Mar., Apr., May & June. Ser. 3a. Vol. 8. Nos. 1, 2, 3 & 4. pp. 83–84.

——. A febre typhoide em S. Paulo.—*Ann. Paulist. Med. e Cirurg.* 1925. Aug. Year 13. Vol. 16. No. 8. pp. 51–71. With 2 charts in text.

CHAND (Wazir); HUNTER (R. N.). Four Suggestive Cases of Enteric Fever.—*Kenya Med. Jl.* 1926. Aug. Vol. 3. No. 5. pp. 146–148.

DUKE (H. Lyndhurst). Observations on the Occurrence of Enteric Fever in Natives of the Uganda Protectorate.—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1925. June 18. Vol. 19. No. 3. pp. 178–179. [1 ref.]

FISCHER (Otto). Eine Schiffsepidemie von Typhus abdominalis.—*Arch. f. Schiffs- u. Trop.-Hyg.* 1925. Oct. 15. Vol. 29. No. 10. pp. 564–572. With 6 charts in text.

NICHOLLS (Lucius) & BURGESS (Edwin). *Bacillus typhosus* morphologically compared with Certain Bacilli present in Natural Waters.—*Ceylon Jl. Sci.* (Section D. Med. Sci.). 1925. Apr. 27. Vol. 1. Pt. 2. pp. 47–56. With 10 plates.

## MISCELLANEOUS.

INDIAN MEDICAL GAZETTE. 1926. Mar. Supplement. 48 pp. **The Indian Medical Year, 1925.**

In the compilation of this critical résumé of multifarious medical achievement in India during 1925 the author acknowledges the assistance of some specialists particularly distinguished in certain subjects. To review a Review of a field that has already to a considerable extent been scrutinized in this *Bulletin* would surely be wasteful and ridiculous excess, but profit may accrue from a survey of its scope, of its merits of method, and of its instructive incidental commentary.

Its scope which is comprehensive, is a revelation of Indian enterprise and successful performance in many important branches of medical research, and the results are marshalled with admirable convenience and conciseness. Subjects are arranged in ten distinct sections, namely : (1) Diseases of the Tropics, their epidemiology, transmission, clinical aspects, treatment, etc. ; (2) Surgery in the Tropics ; (3) Hygiene and Public Health ; (4) Obstetrics, Gynaecology, and Infant Welfare ; (5) Ophthalmology ; (6) Medical Entomology ; (7) Tropical Dermatology ; (8) Pathology, Laboratory Methods, Serology, etc. ; (9) Medicolegal Notes ; (10) Service Notes. In addition there is a "special article" by Major GREEN-ARMYTAGE on recent advances in Obstetrics and Gynaecology, which takes the history far beyond India. In the three larger sections—Tropical Disease, Surgery in the Tropics, and Hygiene and Public Health—each specific disease and each aspect and venue of the subject are dealt with in distinct sub-sections. The section on *Tropical Diseases*, which forms more than three-eighths of the whole Review, consists of 18 sub-sections, including Medical Helminthology, Snake-bite, and Pharmacology. Comment is made (*a*) in the Dysentery sub-section, upon the fact that although five-sixths or more of the dysentery of India is bacillar, all the antiserum required in treatment is imported from abroad ; (*b*) in the Malaria sub-section, upon the insufficient equipment of the Central Malaria Bureau ; (*c*) in the Cholera sub-section, upon the indifference with which the world appears to regard the evidence pointing to the Bengal delta as its chief nidus of the disease ; (*d*) in the Leprosy sub-section, upon a prevalent professional ignorance of the early symptoms and diagnosis of the disease ; and (*e*) in the Pulmonary Tuberculosis section, upon the lamentable absence of any sign in India of a real attempt to grapple with this awful problem. The section on *Surgery in the Tropics* deals severally with Intestinal Surgery, Surgical Tuberculosis, Calculus and Vesical Surgery, Orthopaedic Surgery, Anaesthetics, Antiseptics, and Venereal Diseases and Framboesia ; it also contains miscellaneous notes which include, among other things, the surgery of the ancient Hindus. One of the noteworthy features of the section on *Hygiene and Public Health* is a brief summary of India's new relations to international hygiene.

The chief author of this valuable Review regrets that it is not also a complete bibliographical record. Its price, however, is but one rupee (1s. 6d ) and one does not expect to get 100 oxen for the market price of 9 (ἑκατόμβοι ἐννεαβόιων)

A. Alcock.

Row (R.). **On the Present Position of the Medical Profession and Medical Research in India.**—*Indian Med. Rec.* 1926. Feb. Vol. 46. No. 2. pp. 33–38.

Dealing with the present position of the medical profession in India, the speaker deplored its heterogeneity and heteronomy and lack of cohesion, and consequently its impotence against a general public and governments that—with a few exceptions—are indifferent to the scientific aspects of medicine. As a glaring instance of official indifference, he mentions the abortion—due to the failure of the Bombay Government at the critical moment to supplement private munificence —of the Bombay School of Tropical Medicine.

Speaking of medical research in India he refers to the "five or six Government Research Institutes scattered over the country," but appears to forget the Calcutta School of Tropical Medicine. These Research Institutes, at present very efficiently managed by the Indian Medical Service, must soon suffer from the fact that that service has become unpopular among the higher ranks of the profession. The same argument applies to the Medical Colleges and to the administration of the Indian Medical Research Fund. The prospect, in short, is bad, both for medical education and medical research in India. A gleam of hope is apparent to the speaker in the scholarships of the Rockefeller Foundation and in the blessed word "re-organization"—applied to the Medical Colleges, and in a possible revival of the missionary spirit of "Sacrifice and Service."

A. A.

Reed (Alfred C.). **The Teaching of Tropical Medicine.**—*Jl. Amer. Med. Assoc.* 1926. Aug. 21. Vol. 87. No. 8. pp. 548–549.

A reasoned plea for the teaching of tropical medicine to all medical students in the United States. At Stanford University, San Francisco, there is a course of 22 hours in the department of public health and preventive medicine, supplemented by a laboratory course of the same duration.

A. G. B.

Manson-Bahr (Philip). **Research on Clinical Medicine in Tropical Disease.**—*Jl. Roy. Army Med. Corps.* 1926. June. Vol. 46. No. 6. pp. 442–444.

In this brief article, which forms one of a "Correspondence Circle," Dr. Manson-Bahr writes on protein shock therapy and the treatment of colitis. He has been much impressed with the possibilities of protein shock treatment as applied to the more chronic forms of tropical disease. He employs intravenous injection of a diluted solution of army typhoid-paratyphoid vaccine: the dose, 100 million organisms, is obtained by a 25-fold dilution of the vaccine. The second or third dose must be two or three times this strength. The only tropical disease discussed is climatic bubo, in which the treatment is specific. "It cuts short the process of suppuration and subsidence of the glands takes place almost instantaneously and, in short, the treatment is terminated within a week, whereas formerly the case lingered on for six weeks, two months, or more." Other cases treated with success are, obscure and fugitive

forms of arthritis, arthritis and urticaria and, combined with specific treatment, obstinate tertiary syphilitic lesions. The shock produces high temperature and a leucocytosis.

The second part deals with yatren. This drug, which he finds most efficacious in early infections, he continues to use in amoebic dysentery. In chronic cases, with passage of cysts, he combines the intrarectal injection of 200 cc. of yatren, to be retained as long as possible, with E.B.I. in 3 gr. doses by the mouth; the injection is preceded by thorough lavage with 2 per cent. sodium bicarbonate solution. A ten days' course is usually sufficient. In three cases of acute bacillary dysentery (Shiga) an almost instantaneous improvement occurred after injection of yatren. In mucous colitis also the drug is of value and its use was followed by healing of the ulcers and general improvement in one case of ulcerative colitis.

A. G. B.

KHARTOUM. **The Kitchener School of Medicine, First Report 1924-1925.** [ATKEY (O. F. H.), Director, Sudan Med. Service, President School Council.]—50 pp. With 4 plates & 1 plan.

In a previous number of this *Bulletin* (Vol. 21, p. 309) an outline of the proposed Kitchener Memorial Medical School was sketched. We have here the first report, which contains a historical note and the inaugural address by Sir Lee STACK, with details of the aims, the buildings and administration of the School accompanied by suitable illustrations.

A subscription list was opened in 1916, and £13,000 was raised, all but £2,000 by natives of the Sudan. In May, 1922, building operations commenced, and the school was opened in February, 1924. It is estimated that for annual maintenance a sum of £3,000–£4,000 will be needed or an Endowment Fund of £70,000. The three main objects are: (1) to build up a staff of Sudanese doctors to work under the direction of highly trained British doctors; (2) to associate the educated natives of the Sudan in the development of their country; (3) to provide post-graduate courses for doctors trained at the School. The medical curriculum will at first occupy four years, to be followed by a year spent at a larger hospital; later, another year will be added. The post-graduate classes will be established about 1935.

The organizers realize the vital importance of systematic campaigns against the endemic diseases which are crippling the health and efficiency of the people and interfering with their natural increase; for this a greatly increased staff of doctors is needed. At present, in addition to the senior posts (British doctors), there are 73 junior posts held by Syrian doctors and 33 subordinate posts held by Sudanese assistant medical officers, empirically trained. Both these classes will be replaced by Sudanese doctors. It is proposed to pass out six doctors a year, from January, 1928, a conservative start.

The buildings are described with plan and elevation. The students are drawn from the final year students of the Gordon College. The report of the first year shows that ten students were admitted, and nine reached the required standard in the examination at the end of the year (physics, chemistry and biology).

The Report contains a syllabus of lectures and a list of subscribers; it is anticipated that £6,000 will be needed in 1927.

A. G. B.

OLIVIER (J.). Service médical du Ruanda-Urundi. (Territoire à mandat.) Rapport annuel pour 1924. [**Annual Report of Ruanda-Urundi Medical Service.**]—*Ann. Soc. Belge de Méd. Trop.* 1926. July. Vol. 6. No. 2. pp. 115–144.

The author gives notes about the diseases met with in Ruanda-Urundi in the year under review. Sores and phagedenic ulcers are very common in the natives and very difficult to cure, which is attributed to the bad general state of the population and the frequency of syphilis and yaws. While broncho-pulmonary affections are mild and comparatively infrequent, intestinal affections are severe—amoebic dysentery, ancylostomiasis, flagellate dysentery, bilharziasis and chiefly in the Usumbura plain. Here 92 deaths out of 199 were due to these causes in the order given, 52 being due to amoebic dysentery [it is not stated whether this diagnosis was supported by the microscope]; they are often present in association. Organic affections of the heart are fairly common and are attributed to syphilis and alcoholism, which is general. Of leprosy, 124 cases were recognized, half in Usumbura. Sleeping sickness is considered elsewhere [this *Bulletin*, Vol. 23, p. 898]. There is little information about malaria, but it is stated to be resistant and deadly; five deaths occurred in hospital among 44 patients. Relapsing fever is fairly common but seldom fatal; convalescence is often very long. Smallpox was not seen, but "a few cases of varioloid." A campaign against it was started in 1921, and in 1924 100,000 natives were vaccinated. Yaws is the chief endemic disease, particularly in Urundi. The author regards yaws as a disease which leads to the degeneration and enfeeblement of the race; 15,610 curative injections were given in the year 1924.

Under the heading special hygienic situation of certain groupings, the author considers in turn troops, labourers and prisoners. Among 635 soldiers and their wives and children, a community of about 1,100, there were 5,217 cases of illness, a high figure. The chief causes were wounds and ulcers, pulmonary affections (mild), and intestinal affections, from which one in every 22 suffered. There were 59 cases of bilharziasis [presumably all rectal] in Usumbura among an effective of 468; this high percentage is attributed to a bathing pool supplied from an infected river; the pool has been condemned. Other diseases from which the troops suffered were venereal, seasonal conjunctivitis, malaria, relapsing fever; 1 in 11 suffered from the two last named. A tabular statement is given.

Figures for native labourers show that the intestinal affections, ancylostomiasis, amoebic and flagellate dysentery, and bilharziasis are the chief causes of morbidity and mortality, and that among approximately equal numbers these diseases were about half as prevalent as among the troops. This is attributed to the facts that the soldiers at Usumbura are camped near an infected river and that the labourer is a countryman, whereas the soldiers come from all parts of the Congo.

The prisoners furnish the highest percentage of disease. Their conditions of dietary and work are not inferior to those of the troops and labourers, they are satisfactorily housed and cloth is served out for garments, but the lack of liberty leads to mental depression which impairs their resistance. Moreover, the native of this part of Africa is not used to regular work, and is accustomed to fermented liquors, which form a part of his nourishment and of which he is deprived in prison. A tabular statement shows that among 232 cases of disease

occurring at two prisons there were 75 deaths (influenza, 27; bronchopulmonary affections, 22; "misère physiologique," 7; intestinal affections, 6; etc.). The details of the ration are given [but it is not stated whether the prisoners could supplement it, as men of the other categories doubtless could].

A. G. B.

LE BOURGO. Notes médicales recueillies pendant la campagne de la "Bellatrix" dans l'Océan Indien (1923–1925). [**Medical Notes made during the Voyage of the "Bellatrix" in the Indian Ocean.**]—*Arch. Méd. et Pharm. Nav.* 1926. July–Sept. Vol. 116. No. 3. pp. 197–209. [1 ref.]

A few points of interest may be mentioned. The despatch boat "Bellatrix" called at some of the islands of the Comoro archipelago. At Mayotte there has been a hospital since 1851. Here Dr. KÉRÉBEL has been working on leprosy. In the Comoros and in Madagascar there a dermatomycosis recalling pityriasis, and designated by the Malagasies "Hody potsy"; it consists of patches found chiefly on the face and neck, and has been described by FONTOYNONT (this *Bulletin*, Vol. 20, p. 279). KÉRÉBEL believes that it goes on to tropho-neurotic mutilations and is then confused with leprosy. Of 314 interned persons, he believes 191 to be lepers, 75 to be certainly the victims of this mycosis, and 48 doubtfully so. The distinction is based on clinical data. In Mayotte, as in the other islands, malaria is endemic; filariasis with manifestations in the scrotum and lower limbs is seen and tropical ulcers. Yaws is widespread; family epidemics are not unusual. Gonorrhoea owes its diffusion to the strongly held belief that rapid cure follows sexual relations with a woman of very black skin. Great Comoro also is invaded by malaria, which was quite unknown at the time of the French occupation (? 1886); the medical service is carried on by Hovas, pupils of the Tananarive medical school. Malaria is severe in Réunion. Here are hydromineral stations which are described. The "Bellatrix" called also at Lorenzo Marques, where we learn that a hospital is being built which will contain 1,200 beds, at Mombasa, where smallpox was prevalent, and at Jeddah. The paper opens with remarks on plague in Madagascar.

A. G. B.

WESTPHAL (Carl). Beiträge zur Medizin der kolumbianischen Westküste. [**Medicine on the West Coast of Colombia.**]—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.* 1926. Vol. 30. No. 1. pp. 226–232. [1 ref.]

A sketch of the diseases met with in the south-west corner of the republic of Colombia, the State of Tumaco, in which the author practised for five years. It is a hot, damp land with abundant rain and an average temperature of 27° C., inhabited chiefly by negroes and cross-breeds who live on the coast and along the rivers; away from water is impenetrable marsh forest. *Malaria* abounds and makes it impossible for colonists to settle on the land. Malarial cachexia does not occur and the author has seen only four cases of blackwater. *Yaws* is almost as common, and occurs chiefly in childhood. Judging from the account, all the manifestations described in other countries are seen here. Loss of fingers and toes and deformities are common, and the

author says that early in his career he reported a case to the authorities as leprosy which he later realized to have been one of yaws. Gangosa is not uncommon and is regarded as yaws. *Syphilis* occurs on the coast and is mild; in the uplands where yaws is not seen syphilis is severe.

Compared with malaria and yaws, other diseases have little importance. *Relapsing fever* is present, but is often regarded as malaria; in the author's own practice cases of fever which did not respond to quinine yielded to neosalvarsan. Notwithstanding the primitive character of the hygiene, *enteric fevers* are unimportant. Though the mosquito carrier is found, yellow fever is not observed. *Dysentery* is always present and liver abscess not infrequently. Intestinal worms do not produce symptoms of importance. The skin affection known as *Mazamorra* is frequent, but has no connexion, as is stated in text books, with ancylostomiasis; the author has himself had it. It affects the boot-wearers, seldom the barefoot natives, and is readily cured by application of silver nitrate solution. Leprosy is never seen on this coast.

A. G. B.

VIANNA (Luiz). Nosologia da zona norte do Estado do Ceará. [**Disease in the Northern Districts of Ceará.**]—*Arch. Brasileiros de Med.* 1926. June. Vol. 16. No. 6. pp. 275–276.

Syphilis is very rife. In 1923, of 1,500 school-children examined 70 per cent. showed signs of hereditary syphilis. Miscarriages also are very frequent. As regards yaws, the author mentions one place in the Ibyapaba Hills where practically every one is infected, but in Northern Ceará it is quite rare.

Helminthic infestations are found in at least 70 per cent. of the agricultural labourers. [The nature of the infections is not stated.] Malaria is widespread, especially at the end of the cold months, but this year in the district of Granja it has been troublesome through the cold weather also. Leprosy is uncommon in Ceará itself, but Sobral and Acarahú are endemic foci. Paratyphoid fever is frequent in the northern part of the State, and in 1919–20 there were definite epidemics. Tuberculosis is comparatively rare, except in the town of Granja, where there are many cases. The author questions whether yellow fever exists in the district now. Aëdes is present and there are large numbers of young children dying from "gastro-enteritis" in epidemic form. These are the two bases for the idea that yellow fever prevails, but there is no proof that the gastro-enteritis is due to yellow fever, nor that there is any connexion between the prevalence of this and the existence of *Aëdes argenteus.*

H. Harold Scott.

NÚÑEZ (Solón). **The Occurrence and Non-Occurrence of Certain Diseases in Costa Rica.** *Amer. Jl. Trop. Med.* 1926. Sept. Vol. 6. No. 5. pp. 347–356.

The information is sketchy, and little idea can be got of the relative prevalence of the diseases named.

Enteric fevers have decreased with hygienic precautions and a free vaccination service. There are other forms of paratyphoid beside A and B. The agglutination reaction has not the same diagnostic

value as in other countries and haemoculture is advised. Of leprosy there are 100 known cases. Tuberculosis is mild; a sanatorium exists with provision for 150 patients. In two instances the organism of undulant fever has been isolated. For smallpox vaccination is obligatory; the last epidemic occurred in 1899; two cases of "alastrim" were seen last year. Of scarlet fever there were in 1920 40 cases. Measles is prevalent at intervals; in the last epidemic the most frequent complications were intestinal. Malaria is endemic over two-thirds of the country, and not only on the coast; subtertian is the most prevalent form. The mortality from amoebiasis in the last ten years is given as 2,552 persons or 2 per cent. of the whole mortality. Yaws is believed to occur. Some cases of tropical ulcer have been shown to be leishmaniasis. The information about metazoan diseases is vague. Practically all children over a year are said to harbour ascaris, and adults uninfected with trichuris are rare. Half the population have ancylostomiasis; in 300 specimens Necator only was found.

The diseases noted as absent are: typhus, cholera, plague, rabies, yellow fever, relapsing fever and trypanosomiasis, but of the last it is stated that *Triatoma megista* is common and bites man, and that it harbours *T. cruzi*.

A. G. B.

NICOLLE (Charles). Rapport sur une mission scientifique en Argentine. [**Report on a Scientific Mission to the Argentine.**]—*Arch. Inst. Pasteur de Tunis.* 1926. June. Vol. 15. No. 2. pp. 166–172.

A brief report of two months spent in the Argentine Republic on the invitation of the President of the National Department of Hygiene. The author was able to take from Tunis the virus of typhus in guineapigs and that of kala azar in dogs for study and maintenance in the Argentine laboratories. The scientific results of the mission will appear later.

A. G. B.

MÜHLENS (Peter). Beiträge zur Pathologie von Südamerika. [**The Pathology of South America.**]—*Beihefte z. Arch. f. Schiffs- u. Trop.-Hyg.* 1926. Vol. 30. No. 1. pp. 143–160. With 17 text figs.

This "preliminary communication" goes over the same ground as a paper published in the Proceedings of the Conference of the United Fruit Company [this *Bulletin*, Vol. 22, p. 620]. Some good photographs are added. A detailed report of the expedition will be issued shortly.

A. G. B.

NILES (George M.). **Afro-American Therapeutics.**—*New Orleans Med. & Surg. Jl.* 1926. May. Vol. 78. No. 11. pp. 747–749.

The author writes from Atlanta, Georgia, after 35 years of practice in the South, and his conclusions apply to "quadroons and those of darker hue." He finds that for the Afro-American the dose of a cathartic is 50 per cent. greater than for the Caucasian. The same applies to emetics, but if the action required is on the vomiting centre less is needed; e.g., 25 per cent. less apomorphia. The negro requires some 30 per cent. less anodyne for the relief of pain, and less sedative, such as bromide and trional. Counter-irritants, however, must be applied to the negro in considerable strength to produce results.

As a result of the study of 128 cases in which the stomach was unaffected and a test meal was given the author believes that "the normal free hydrochloric acid in the Afro-American exceeds that in the Caucasian stomach by at least 6—probably 10 per cent."

A. G. B.

AMERICAN MISSION HOSPITAL, Petchaburi, Siam. **Annual Report 1925-1926.** [NEDERGAARD (Niels).—15 pp. [No Printer or Publisher.]

A recent report from this hospital was noticed in Vol. 23, p. 212. The number admitted as in-patients during the year under report was 216, as out-patients 1,096; 1,734 laboratory examinations were made, of which 869 concerned the blood and 486 the faeces. Ancylostomes were detected in 80 cases, ascaris in 49, trichocephalus in 41.

Institutions under native direction are now doing similar work and it is proposed that the hospital service be diverted to health service, which is defined as obstetric work in the homes, instruction in care of home and children, instruction given on tour in the prevention of disease and the treatment of minor ills, advice to "local medical people" in diagnosis and treatment.

A. G. B.

IKEDA (Kano). **The Blood in Smallpox during a Recent Epidemic.**—*Arch. Intern. Med.* 1926. May 15. Vol. 37. No. 5. pp. 660–673. With 5 text figs. [7 refs.]

Of 480 cases admitted to the Minneapolis General Hospital, including 191 discrete pustular cases, 110 confluent pustular, 131 haemorrhagic pustular and 48 purpuric, the author examined the blood at different stages in "approximately 200" [how many of each is not stated]. The difference between the blood findings of the frank pustular forms and the purpuric form were "strikingly constant," and are shown in a series of curves. The summary and conclusions seem of sufficient importance to be given in full. The author refers to HOFFMANN's work [this *Bulletin*, Vol. 21, pp. 332–3] and suggests that the eosinophilia recorded by him was due to prevalence of intestinal parasites, or a peculiar tissue reaction of the natives of Cuba to the smallpox virus.

Summary.—"An analysis of approximately 250 examinations of the blood in all forms of smallpox reveals the following important points:

"1. The blood of the pustular forms of smallpox differs essentially from that of the purpuric form. This is definite and constant and is of value in differential diagnosis.

"2. The platelets are greatly diminished in the initial period in all forms of smallpox. In the pustular forms the greatest diminution is reached in the vesicular stage, followed by a rapid rise through the pustular stage into the desiccation period. In the purpuric form the platelet count shows a consistent downward course until death.

"3. The total leucocyte count shows at the onset an increase, as a rule, in proportion to the severity of the subsequent course of the disease. In the mild pustular forms a definite leukopenia is present in the maculopapular

stages. In the severe and fatal pustular forms a high total count is maintained throughout the course of the disease. Its maximum is reached during the early part of the desiccation period. In the purpuric form an initial hyperleukocytosis is steadily maintained. This is considerably higher in the primary type than in the secondary type. There are few exceptions, both in the pustular forms and in the purpuric form, in which a leukopenia or a normal leukocyte count persists throughout the course of the disease.

"4. The polymorphonuclear neutrophil percentage shows a steady downward course in all forms of smallpox except in the desiccation period of the severe and fatal pustular forms. A definite neutropenia of 40 per cent. is recorded during the maculopapular stages in the mild form which shows a slight rise during the subsequent stages, declining again to 47 per cent. in the desiccation period. A definite polynucleosis is present in the initial and the maculopapular stages in the severe and fatal pustular forms. A decisive neutropenia is obtained in many of the late stages of the purpuric form. The neutrophil fall in this form is strikingly sudden and spectacular in the presence of a hyperleukocytosis.

"5. The temperature and the polymorphonuclears run a parallel course while the total leukocyte count shows a reverse curve in all forms of smallpox.

"6. No characteristic changes are noted either in the hemoglobin or in the erythrocyte count. The fact is of significance in the purpuric form, in which there are morphologic and qualitative changes of an advanced anaemia, usually accompanied by demonstrable, profuse hemorrhages. A definite secondary anaemia may develop in severe pustular and protracted secondary purpuric cases, in which a quantitative decrease of the hemoglobin and of the erythrocytes, together with a proportionate morphologic variation, is demonstrated. The decrease may be of such gravity as to require restorative measures.

"7. Morphologic changes in the leukocytes and the erythrocytes indicate a severe stimulation of the bone marrow and, in themselves, merely signify various degrees of toxemia or infection. In the blood of the purpuric form, however, there is a series of characteristic metamorphoses in the mature leukocytes which appears to indicate early degenerative changes, specific only in this form of smallpox, and which has served as the only positive laboratory evidence in its diagnosis. This often occurs before any of the clinical signs are evident."

*Conclusions.*—"The blood of smallpox shows characteristic findings which, if properly interpreted, are of definite diagnostic and prognostic value.

"1. The earlier the rise of the platelets, the sooner the approach of the desiccation period, prognosticating a shorter course of the disease.

"2. A definite leukopenia during the maculopapular stages indicates, as a rule, a mild discrete form. A progressive leukocytosis with an early high polynucleosis predicts a severe form. The higher the values, the more probable the fatal outcome.

"3. The early appearance of normoblasts, basophilic stippling and polychromatophilia, without evident anaemia, is an infavourable sign. It invariably means the purpuric form of smallpox.

"4. Condensation and fragmentation of mature leukocytes are found only in the purpuric form of smallpox. They appear comparatively early in the primary type of purpuric smallpox and are usually accompanied by pathologic normoblasts, basophilic stippling and polychromatophilia, without visible anaemia.

"5. A rapidly progressive, absolute lymphocytosis is a constant characteristic of purpuric smallpox.

"6. Scarlatinal and other exanthems, infectious purpura and toxic rash with petechiae, etc., can be definitely differentiated from the purpuric form of smallpox during its erythematous stage by these blood findings."

A. G. B.

DA ROCHA (Franco) & RANGEL (Mario). Tratamento da variola pelo Xilol. [**The Treatment of Smallpox by Xylol.**]—*Rev. Med.-Cirurg. do Brasil.* 1926. May. Vol. 34. No. 5. pp. 215–217.

During a period of five and a half weeks in March and April of this year there came under the authors' care 145 cases of smallpox, 26 of the discrete form, 20 generalized, 87 confluent, and 12 haemorrhagic. Of this number, 18 of the confluent and 9 of the haemorrhagic died, giving a mortality of 18·62 per cent. The treatment employed was xylol in doses of 20–30 drops for children, 80–120 for adults, given in water, milk, or wine, thrice daily. If administered in the early stages it appeared to prevent suppuration ; in the pustular stage it caused a drying up of the pustules, and it lessened or prevented scarring. It is practically ineffectual in haemorrhagic cases or in confluent forms in debilitated subjects.

The use of this drug in variola was first recommended by ZUELZER in 1871, and its reputed benefits found confirmation from WICHNEWSKY in 1903, and BELIN in France in the following year.

H. Harold Scott.

KADANER (M.). Trois cas de tétanos à Stanleyville. [**Three Cases of Tetanus at Stanleyville.**]—*Ann. Soc. Belge de Méd. Trop.* 1926. Jan. Vol. 5. No. 2. pp. 125–126.

A short account of three cases of tetanus in adult natives, said to be the first observed in the Stanleyville locality. All were fatal. In two instances infected wounds were present, in the third there was dysentery and no external wound ; the question is raised whether the point of entry was a dysenteric ulcer or the attack allowed the development of bacilli which had previously entered by a breach of skin. [Anti-tetanus serum is not mentioned and was presumably not available.]

A. G. B.

HEALTH. Melbourne. 1926. Jan. Vol. 4. No. 1. pp. 28–29.—**Commonwealth Serum Laboratories. The Serum Treatment of Tetanus.**

Of eight cases of tetanus in children treated at Brisbane with "Commonwealth" serum only one died. The average incubation was 10¼ days in six cases in which the period could be ascertained and in two of these only seven days. On admission 90,000 units were injected, 75,000 intravenâ and the rest in muscle, repeated as a rule in this or smaller dosage on the two succeeding days. The patients who recovered received from 120,000 to 330,000 units. All but one were between 5 and 9 years of age.

A. G. B.

LHUERRE (H.). Sulfate d'atropine et sérothérapie dans le traitement du tétanos. [**Atropine Sulphate and Serotherapy in the Treatment of Tetanus.**]—*Bull. Soc. Path. Exot.* 1926. Jan. 13. Vol. 19. No. 1. pp. 66–69.

The author has been in charge of the Infectious Diseases part of the Native Hospital at Dakar for four years and has had many cases of tetanus under his care. Treated in the usual way 77 per cent. died.

Experience with plague has shown that the West African native tolerates injections of serum in his veins up to 100 and 150 cc. He therefore gave 100 cc. of antitetanus serum in one dose daily by vein till the symptoms of spasm and hyperexcitability were clearly diminished, and to protect the muscles served by the phrenic, glossopharyngeal and pneumogastric nerves from the influence of the tetanic toxin he used belladonna, with bromide, chloral and morphia. Later, for the belladonna he substituted a hypodermic injection of sulphate of atropine. Since he has employed large doses of serum intravená and atropine he has had 16 tetanus cases, of which 12 have recovered.

A. G. B.

NORMET. La "Bouffissure d'Annam." **Annam Swelling.** *Bull. Soc. Path. Exot.* 1926. Mar. 10. Vol. 19. No. 3. pp. 207–213. With 1 text fig.

This name is given to a condition in which the patients have oedema, more or less general, of the cellular tissue and dropsy of the serous cavities, especially the peritoneal. Other cardinal symptoms are loss of flesh with muscular wasting and anaemia. Cardiac or renal symptoms are absent. There are no nervous symptoms, which puts beriberi out of question, but the disease is probably one of deficiency. The patients are workers on the land or coolies; persons in easy circumstances are never affected, nor are the coastal inhabitants who live on fish. Cases occur through the year, but are especially numerous in times of food scarcity. At first glance the victim appears fat and well liking, but closer inspection reveals the underlying emaciation which is most prominent at the shoulders and upper part of the chest. He is dulled, replies slowly to questions, his mouth is usually open and his gaze vacant. The anaemia may be such as corresponds to 600,000 red cells and 10 per cent. haemoglobin, or is less extreme. The diagnosis is confirmed by the examination of the blood and urine for urea; in Annam swelling the amount in each is diminished. The urea excreted in the urine may be 8 gm. in 24 hours, and that of the blood may not reach 25 cgm., or these figures may be considerably less. Another less important symptom is arterial hypotension, and yet another a peculiar dryness of the skin.

At autopsy the organs are extremely pale, and fluid is found in all the serous cavities. To gross appearance and under the microscope, the organs are normal. Most of the patients have intestinal parasites, but these infestations are very common in Annam. Administration of chloride of calcium drives away the oedema, but does not avert death. Overfeeding gives no result; the food is not assimilated. The author therefore determined to inject a nitrogenous substance in the most assimilable form, that of amino acid, and choose glycocoll as of moderate price and borne well in large doses. BROCHA and FREDERICQ have demonstrated its vaso-dilator action in small doses. Experience has indeed shown that small doses cause the oedema of the tissues and serous cavities to disappear and that the drug acts like a catalyser on the assimilation of nitrogenous substances. A daily dose of 20 cgm. for ten days in subcutaneous injection rids the patient of the oedema and improves his assimilation. After a week, and then after a fortnight, the course is repeated. The formula is Glycocoll 4 gm., salt solution ·8 per cent., 1,000 gm. Formerly, whatever the treatment,

the patient was carried off sooner or later by profuse diarrhoea, or progressive cachexia, or syncope; the mortality was 95 per cent. Now recovery is almost invariable in a few weeks. A photograph represents a boy (1) swollen with the disease; (2) emaciated after the loss of the fluid, and (3) after recovery of subcutaneous fat. The appetite is by then large, and it is safe to overfeed. In two months the

Annam Swelling—left, on arrival at the hospital; centre, after disappearance of oedema and ascites; right, recovered.

[Reproduced from the *Bulletins de la Société de Pathologie Exotique.*]

appearance is normal. For the anæmia, which is slow in disappearing, citrate of soda and "tartrate ferrico-potassique" in intravenous injection are used. With recovery the urea in both blood and urine increases. In some instances, in addition to the glycocoll treatment, it is necessary to tap the peritoneal cavity. It is stated that the disease has thousands of victims among the agricultural population, with whom rice constitutes almost the sole food.

[One is reminded of epidemic dropsy and famine dropsy. A similar condition was described by BIGLAND from East Africa (*Lancet*, 1920, Vol. 1, p. 243; this *Bulletin*, Vol. 14, p. 76). In famine dropsy the pulse is slowed. A symptom which does not find mention here.]

A. G. B.

KÉRANDEL (J.). Sur la bouffissure d'Annam et sa pathogénie. **Annam Swelling and its Pathogenesis.** *Bull. Soc. Path. Exot.* 1926. Apr. 14. Vol. 19. No. 4. pp. 302–310. [1 ref.]

Kérandel agrees with NORMET that this disease is both frequent and serious in Annam; it is the chief cause of mortality at the hospitals at Hué and elsewhere. There have long been local controversies about it, some observers attributing it to ancylostomiasis, others to malaria or beriberi. THIROUX in 1920 [see this *Bulletin*, Vol. 17, p. 238] attributed the syndrome to the association of ancylostomiasis and malaria with syphilis; he entitled his paper, "Grave Anaemia following multiple Infections in Annam," and states that 95 per cent. gave a positive Bordet-Wassermann. Kérandel is inclined to follow THIROUX and stresses the significance of hereditary syphilis.

The swelling may be slight or so extreme that "the patient appears bloated like a leather bottle." The anaemia is very variable, its grade corresponding with that of the ancylostome infection. Ascites was seen in 30 per cent. of [an unnamed number of] cases; in its absence there is always some distention and relative dulness of the abdomen, which is an early sign. In 77 per cent. of cases the liver was diminished in volume, the spleen hypertrophied. Besides the diminution of urinary urea reported by NORMET, there is a reduction of chlorides and oliguria. Other symptoms are dulness and extreme emaciation.

Five autopsies have been made, but have revealed nothing striking. They confirm the atrophy of the liver, which is of yellowish, earthy, or "dead-leaf" colour. There seems to be a tendency to fibrosis of the viscera. The mesentery, when the patient dies with oedema, is very oedematous. Few microscopic examinations are on record.

The great majority of the patients harbour numerous ancylostomes, but this does not explain every case; in four, none was found in repeated examination of stools. A table of 29 cases, showing the sex and age, occupation, number of ancylostomes (in decreasing order), haemoglobin value, W.R. and degree of swelling, shows that whereas the anaemia varies with the degree of ancylostome infection, the oedema does not; general anasarca occurred in several cases with 30 worms or less, and relatively high haemoglobin values. Nor is the oedema attributable to malaria. The disease may occur in patients from the delta where fish abound, and in seasons of plenty as well as scarcity; he agrees that it is rarely met with in the well-to-do. It is most frequent between the ages of 12 and 31, rare above or below these limits; many subjects are about 20 years. This limitation of age would, in his opinion, be against a dietetic origin. Signs of inherited syphilis are found in exceptional frequency in these patients. In 51 cases the serum reaction was tested by one or two methods; the percentage of positives was 83, though the serums of other patients, in whom syphilis was suspected, examined at the same time, gave the smaller percentage of 60.

The author says that the results of treatment tend to confirm the importance of the part played by syphilis. Mercury was badly tolerated and "914" was given. The general state improved, but it appears that 20 per cent. died either in hospital or after removal.

In the discussion which followed M. LEGER mentioned other parts of the French Colonial Empire where ancylostomiasis, malaria, and syphilis abound but this curious disease is absent. MOTAIS said that he took part in the study made by THIROUX and that most of the patients recovered with thymol, quinine, "914," diuretics, and a milk diet; he believed, however, that some part of the aetiology remains undiscovered. GAUDUCHEAU said that the limitation of age was not against a food deficiency origin, because the physiological needs of the organism vary with age and are especially great in the growth period. "In these countries," he said, "doctors ought to interest themselves more than they have done up to the present in the diet of their patients, draw up complete food balance sheets and make sure that the local dietaries really correspond to the needs of the economy."

A. G. B.

HINGSTON (H.). **Epidemic Dropsy and Secondary Anaemia of Pernicious Type.**—*Indian Med. Gaz.* 1926. Aug. Vol. 61. No. 8. pp. 373–375. [1 ref.]

This is a remarkable record from the Medical College Hospital, Calcutta, which must be given in some detail.

A Mohammedan male was admitted on January 8th, 1926, complaining of swelling of the legs, general weakness and shortness of breath, difficulty of walking owing to stiffness of the legs, and failing vision. On examination the abdominal wall and scrotum, as well as the legs, were found to be swollen. The heart was dilated with a systolic murmur and pulse 120. The R.B.C.s were 900,000; haemoglobin 25 per cent.; colour index 1·39; marked poikilocytosis, many normoblasts and a few megaloblasts, W.B.C.s 4,300; Wassermann negative. No enlargement of glands or spleen. Marked spasticity of legs and arms. Deep reflexes greatly increased with sustained ankle and patellar clonus; plantar reflexes flexor. No sensory loss or hyperaesthesia. The ophthalmic surgeon reported massive round haemorrhages of both retinas.

The patient related that on September 21st, 1925, he visited relations in the Jessore District, a household of 22 persons. On October 6th rice brought from a river-side village caused diarrhoea, and after a few days was returned, but on October 13th the servant whose duty this was could not go because his legs were swollen. On October 29th, at which date there had been no other cases of illness, the patient returned to Calcutta, and a few days later had to take to bed owing to swelling of his legs and face. He returned to the Jessore village on November 30th and found that five inmates had died of "swelling" and all the rest were ill, with weakness, anaemia, and, in some instances, stiffness of legs and failure of vision, following the swelling. By the end of December, 15 of the 22 were said to have died. The patient himself received iron and arsenic treatment in hospital, rapidly improved and left on March 3rd.

On February 20th his brother was admitted with the same train of symptoms, including retinal haemorrhages and spasticity. He also improved under treatment. Two other members of the family were examined, one of whom still had retinal haemorrhages.

Major Hingston discusses this "family tragedy." He thinks that the primary disease was epidemic dropsy, and that it was due to infection of the rice consumed, but can find no record of pernicious anaemia as a sequela. The cases seem to show "the continued action of a primary acute infection" which the author suggests is an indication of the causation of many of the acute anaemias that are common in Bengal. The spasticity he would explain by small haemorrhages in motor tracts of the cord, such as were seen in the retinas.

Major Hingston notes that the tragedy did not create any unusual excitement, and would not have been heard of if one of the victims had not been a Calcutta resident and an educated man. It is probably not an isolated occurrence.

A. G. B.

De (S. N.). **Epidemic Dropsy.**—*Indian Med. Rec.* 1926. July. Vol. 46. No. 7. pp. 193–195.

An account of a small epidemic in 1919 in 76 Bengali Hindus. These were members of 13 families, and intervals of 3-5 days were noted between the first and subsequent cases; 32 per cent. of the cases were in children of 5-10 years. The symptoms were anorexia, malaise, palpitation, dyspnoea, oedema of lower extremities in all cases; fever in 63 and gastro-enteritis in 53. Rich and poor were equally affected; samples of rice collected were of varying grade from the coarsest to the best. The author attributes the condition to toxins, rather than food deficiency, toxins elaborated in the digestive tract owing to "faulty metabolism of protein in general and rice in particular." Where members of two families were attacked they were in frequent and close contact.

A. G. B.

Archibald (R. G.). **A Case of Sickle Cell Anaemia in the Sudan.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1926. Jan. 21. Vol. 19. No. 7. pp. 389–393. With 4 figs. on 1 plate. [7 refs.]

So far sickle-cell anaemia has been recorded only in America, in negroes (see this *Bulletin*, Vol. 20, p. 670, and Vol. 21, pp. 670, 671, and 926) and a mulatto (see this *Bulletin*, Vol. 7, p. 329). The patient in the present instance was an Arab boy who had suffered since infancy from debility and irregular attacks of fever. The parents were healthy intelligent Arabs, and there was nothing in the family history that had any apparent bearing on the case. The associated symptoms were debility, pyrexia, slight jaundice, oedema of the feet, and enlargement of the liver and lymphatic glands. Blood examination showed, besides the characteristic sickle-shaped red cells, a diminution of the red cells, leucocytosis, low haemoglobin percentage, erythroblasts, and phagocytosis of red cells.

A. A.

Anderson (Horace B.). **Sickle-Cell Anaemia. Report of an Active Case.**—*Amer. Jl. Med. Sciences.* 1926. May. Vol. 171. No. 5. (650). pp. 641–648. With 4 text figs. [8 refs.]

This case occurred in a negro boy, born and reared in Philadelphia. During the first six weeks in hospital the characteristic "sickling" phenomenon was not in evidence; afterwards the red blood corpuscles

on standing for 24 hours showed 90 to 95 per cent. "sickling." The 12 year old sister also showed 50 to 75 per cent. "sickling," but the father appeared to be normal. The mother and a brother who formed the family complement were not accessible.

A. A.

PERUZZI (Mario). Granulomi e granulomatoidi. Ricerche-istologiche e studio sistematico con 3 tavole di microfotografie. [**Granulomata and Granuloma-like Conditions. Their Histology and Systematic Study.**]—*Ann. di Med. Nav. e Colon.* 1926. Mar.–Apr. Year 32. Vol. 1. No. 3–4. pp. 129–186. With 27 figs. on 3 plates. [222 refs.]

This article presents an excellent account of the various granulomata and granuloma-like conditions. The subject is treated in several parts, each with sub-divisions. The first deals with the characters common to granulomatous processes, describing the exudations and the various cells found, their origin and the changes which they undergo. The second goes still further into the histology of the subject, cellular infiltrations, necrotic degenerations and the cytological characters seen in tuberculosis, leprosy, blastomycosis, leishmaniasis and other conditions. In part III the various granulomata are taken up *seriatim*, and for each is given a short account of the general characters and the histological picture, followed by a few remarks on the condition, very apt and to the point. Tuberculosis, syphilis, leprosy, glanders, rhinoscleroma, yaws, cutaneous and muco-cutaneous leishmaniasis, granuloma venereum, verruga peruviana and schistosomiasis are considered under the heading "Infective granulomata," showing that the author interprets the term somewhat widely. A second sub-section treats of the mycetomas—Madura foot, actinomycosis, blastomycosis and sporotrichosis; the third of lymphogranulomata, including climatic bubo, Hodgkin's disease, trachoma and mycosis fungoides. The "granulomatoid" conditions are divided into three groups: the *ulcerous*, of which phagedaenic tropical ulcer is the type, the *vegetating*, which is the same as, or closely allied to, the botryomycosis of animals, and the *nodular*, by which is implied the tuberculoid changes set up by the presence of foreign bodies.

There is an appendix in which the characters already described in moderate detail are recapitulated in a brief form as a sort of diagnostic table. There are three plates of 27 microphotographs very well reproduced.

It is not possible to do more than indicate the scope of this article which, in spite of its length, is far too short to treat so vast a subject adequately. The diagnostic table would be useful as an indication or guide for students at a class lecture or demonstration, but those who are engaged in the study of the question know that the differences are not so clear cut as the author would have us believe (would that they were !). This paper would form an excellent basis for a larger work and might well be amplified to book-form.

H. Harold Scott.

PAWAN (J. L.). **A Case of Cerebro-Spinal Meningitis.** *Ann. Trop. Med. & Parasit.* 1926. June 24. Vol. 20. No. 2. pp. 195–196.

The isolation of the meningococcus in Trinidad and its apparent association with otitis media are the chief points of this paper. Gram-

negative diplococci were found in the cerebro-spinal fluid and were grown on glucose hydrocele agar; serological tests were not made. Death occurred ten days after the patient's admission to hospital; the diplococci were found also in exudate at the base of the brain and in the mastoid cells.

A. G. B.

RILEY (William A.) & WATSON (Cecil J.). **Histoplasmosis of Darling. With Report of a Case originating in Minnesota.** *Amer. Jl. Trop. Med.* 1926. July. Vol. 6. No. 4. pp. 271–282. With 3 text figs. [20 refs.]

A fourth case (in this instance in a German woman who had lived continuously in Minnesota for 42 years) of histoplasmosis, a disease hitherto known only from the three cases reported by DARLING from Panama. In this case as in those the spleen was greatly enlarged, the lungs granulomatous and fibrosed, and enormous numbers of Darling's *Histoplasma capsulatum* were found in the phagocytes (macrophages) with which the lung, liver, spleen and lymph-glands were loaded. The enigmatic nature of this organism, which has variously been regarded as a Protozoon (a Coccidiam, or a Leishmania or a Leucocytozoon), as a yeast, and as a mould, is here briefly discussed, as well as the nature of the disease caused by it, which has been supposed to be identical with epizootic lymphangitis of horses. The authors accept the opinion of ROCHA LIMA that the organism is closely related to *Cryptococcus farcinimosus* of Rivolta (the cause of epizootic lymphangitis, variously considered to be a yeast or a mould), but they incline to think that the disease is not actually identical with epizootic lymphangitis, though its organism may bear a relation to the organism of that disease analogous with that existing between *Leishmania donovani* and *L. tropica.*

A. A.

CADE (A.), MORENAS & RAVAULT (P.). Parasitisme intestinal et tuberculose. [**Intestinal Parasitism and Tuberculosis.**]—*Arch. des Malad. de l'Appareil Digestif.* 1925. Dec. Vol. 15. No. 10. pp. 991–1004.

This is a discourse on a speculative subject. The authors allow that the association of tuberculosis with intestinal parasitism may sometimes be fortuitous, but yet quote impressively statistics given [no exact reference] by L. BERNARD, SALOMON, and MÉLAMET of 40 per cent. of cases observed by them [total number not stated] of pulmonary tuberculosis associated with intestinal parasitism. They themselves produce four cases of combined pulmonary and visceral tuberculosis having dysentery [amoebic and unspecified] as an antecedent, three with Giardia infection as an antecedent, one having strongyloides as an antecedent, and one case of ileo-cæcal tuberculosis coincident with Trichocephalus. They also discuss the inverse aspect of the association —parasitism subsequent to tuberculosis. They discuss again, when the parasitism is antecedent, the influence and line of action of the intestinal parasites; do they prepare the soil for the tubercule bacillus, in a general way? do they form a muster-ground for the bacilli, in the intestine? do they themselves perchance inoculate the bacilli? All these are very familiar questions.

A. A.

MARCOU-MUTZNER. L'antagonisme paludo-tuberculeux; un projet de malariothérapie tuberculeuse. [**Antagonism between Malaria and Tuberculosis. Proposed Malariatherapy.** *Presse Méd.* 1926. Aug. 18. Vol. 34. No. 66. pp. 1052–1053.

It is generally considered, writes the author, that malaria makes the bed for tuberculosis. This is not the case in Corsica, where both infections are common. In the last six years among more than a thousand patients he has not seen the two diseases in association except in ten instances, or about 1 per cent. If they were not antagonistic they would be met with, he thinks, much more frequently in the same patient. Moreover, whereas tuberculosis in Corsica is an acute disease progressing nearly always to death in 3 to 6 months, in the hundredth patient who is infected with malaria it takes a chronic sclerotic form with long periods of arrest. Malaria, on the other hand, is mild in Corsica, and when properly treated is not a serious disease. He proposes therefore to treat tuberculosis by malarial inoculations.

A. G. B.

REYNAUD (Gustave). Le "foie tropical." [**Tropical Liver.**]—*Rev. Prat. Malad. des Pays Chauds.* 1926. Mar. Year 5. Vol. 6. No. 1. pp. 6–21. [3 refs.]

The author discusses and emphasizes the preponderating part taken by the liver in the general body economy, points out that the commonest complaints in Europeans in the tropics are "bowel" ones, and lays stress on the wrong habits, diet, and clothing of the Europeans—especially over-indulgence in alcohol. The consequent ill health is due to a derangement of liver function produced by these misdoings. "Tropical liver" designates a state of hyperfunction and hyperaemia of that organ in which climate plays but a secondary rôle, the "primordial" being played by ignorance and neglect of hygiene. [Amoebic hepatitis is not mentioned—one may conclude therefore that the author excludes this definite and proved entity from his group—"Tropical liver." For the rest—there is as yet no satisfactory test for liver function.]

H. M. Hanschel.

GAEHLINGER (H.). Quelques applications des travaux recents au traitement thermal de l'amibiase et du paludisme. [**Applications of Recent Work to the Spa Treatment of Amoebiasis and Malaria.**]—*Rev. Prat. Malad. des Pays Chauds.* 1926. Feb. Year 4. Vol. 5. No. 10. pp. 460–475.

It has been said that one cannot name an affection of the liver which is not followed sooner or later by intestinal disturbance, just as chronic irritation of the mucosa of the colon rapidly brings about a change of the liver functions. Intestinal amoebiasis and malaria furnish an example of this interdependence. DAVID and SEGAL state this *Bulletin*, Vol. 21, p. 597] that of patients with chronic malaria a larger percentage harbour intestinal parasites, chiefly nematodes, than in the case of other patients. The parasites secrete toxins which cause anaemia, and the blood corpuscles are rendered more vulnerable to the malarial parasite. According to LOEPER in cases of colitis the red cells are diminished in number with a slight haemolytic jaundice and the

anaemia is caused not only by haemolysing microbian toxins, but also by the absorption of cell products, pancreatic and intestinal. If there is also hepatic insufficiency, there is every inducement for the multiplication of plasmodia, and treatment must have regard to each element of the symptoms. Similarly LE NOIR and Mathieu DE FOSSEY (*loc. cit.* Vol. 19, p. 688) have shown that the surest way to manifest a latent amoebic dysentery consists in the administration of biliary extracts in increasing doses till diarrhoea appears; motile forms of the amoeba are then seen. The same result can be attained by stimulation of the biliary secretion as by the administration of Chatel-Guyon water. Lamblia has been shown to harbour in the duodenum and LIBERT has found haematophagous amoebae in the same situation, so that "the hepatic function reacts on the course of amoebiasis."

These observations are then applied to the treatment of malaria and amoebiasis at Chatel-Guyon.

A. G. B.

PANAYOTATOU (Angelica). **Cystitis caused by** *Amoeba histolytica* **and** *Schistosoma mansoni.*—*Brit. Jl. Children's Diseases.* 1925. Oct.-Dec. Vol. 22. Nos. 262–264. pp. 289–291.

Dr. Panayotatou records the case of an Egyptian boy at Alexandria, aged seven, who had passed blood in the urine for four months. There was no history of dysentery. After centrifugation of the urine there were found a large number of amoebae and ova of Bilharzia of the *S. mansoni* type. The amoebae were 12–22 "mm. in size" and showed granular endoplasm and translucid ectoplasm. No amoebae or ova were found in the stools after repeated search. Emetine treatment led to complete recovery.

A. G. B.

SOETEDJO. Over het voorkomen van rheumatische hartaandoeningen in Nederlandsch-Indië. [**The Occurrence of Rheumatic Affections of the Heart in the Dutch East Indies.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1926. Vol. 66. No. 2. pp. 236–241. [11 refs.] With 3 figs. on 1 plate.

Aschoff's tubercles in the heart muscle, consisting of large cellular elements, with large, often polymorph nucleus, round an apparently necrotic centre, are considered to be pathognomonic for rheumatic affections. In 20 cases of endocarditis in the Dutch East Indies the author found these units only once, in a native girl of about 16 years. This was the only instance in a series of 2,300 microscopical examinations of hearts after death from natural causes. These facts prove that real rheumatic infections are extremely rare in the D.E.I.

W. J. Bais.

VAUGHAN (Kathleen). **Osteomalacia in Kashmir.**—*Brit. Med. Jl.* 1926. Mar. 6. pp. 413–415.

Osteomalacia, the author writes, is extremely common in Kashmir. During one of the three years in which she was medical superintendent of the Zenana Hospital at Srinagar, of 29 Caesarean sections 25 were

due to pelvic deformity from that cause. Almost all Kashmir women who have borne children are affected in some degree, with the exception of the boatwomen, who live on the Jhelum river, are out in all weathers, take raw vegetables with their rice, and drink the milk of the cow or goat that is kept on board. "The disease is unknown among men in any part of the world" [others state that it is very rare in males]. It is worse in winter and early spring during and after confinement to the house. A common history is, confinement to the house at 8 or 9, marriage at 10 or 11, menstruation at 12 or 13, and restriction to the house till the first child is born. Lactation is continued in Kashmir from 2 to 4 years. The disease usually begins with the first pregnancy, or soon after marriage.

The most obvious symptoms are pains in the bones, ribs, pelvis and shafts of the long bones, which are tender on pressure about the centre. The epiphyseal lines seem to be quite unaffected and rickets is rare in Kashmir. Tetany is a common accompaniment. Many of the males of osteomalacia families suffer from tuberculosis, "too constant a relationship to be mere chance." Two indigenous cures for "trouble in the bones" are: a special fireclay, which analysis showed to contain 16 per cent. of calcium phosphate and other minerals; fish liver pills. The author found that out-patients given cod liver oil and sent out on the lake in open boats improved rapidly.

The details of dietary are meagre. The richer classes cook all their food and no raw vegetable or fruit is eaten. The water is usually contaminated. In the winter the women go out as little as possible, live in the lowest rooms, where the windows are very small and are often closed by wooden lattice work; some rooms have no windows, but "that the light supply is sufficient for health in all ordinary life is proved by the rarity of rickets." Hills overhang the city of Srinagar, and in the winter a thick pall of smoke and fog. The importance of light is shown by the statements that sunlight alone can cure the disease, and that whereas among Afghan women at Peshawar who live an open-air life the disease is unknown, cases are reported in Hindu shopkeepers' wives who were kept in purdah; again, in Bombay the Parsee women are free, but the weavers, who live in dark rooms behind the looms, suffer severely [see also this *Bulletin*, Vol. 22, pp. 977–9].

A. G. B.

GARDNER (Fletcher) & BROWN (Aubrey K.). **Typhus Fever along the Rio Grande in the United States in 1924 and 1925.**—*Milit. Surgeon.* 1926. Feb. Vol. 58. No. 2. pp. 165–169.

The authors refer to the epidemic studied by SINCLAIR & MAXCY, though they do not give a reference to their paper [see this *Bulletin*, Vol. 22, p. 656]. In all cases under observation clothing, bedding, and bodies were found free from lice, and in a U.S. Army Post no lice were ever found. They suggest that transmission may have been effected by fleas. Two cases are described; both gave a Weil-Felix reaction, which the authors consider positive if agglutination occurs in dilution greater than 1 : 40. The only point not noted in the previous paper is "the typical odor, which resembles that of musty straw." [SINCLAIR & MAXCY found head lice in 12 of 20 families in which cases occurred and regard the head louse as the probable vector.]

A. G. B.

DESTEFANO (Francisco) & VACCAREZZA (Raúl F.). Tratamiento del carbunclo humano. Resultados comparativos de diversos tratamientos sobre 414 casos de carbunclo. Suero anticarbuncloso. Suero normal. Inyecciones de peptona. **Treatment of Malignant Pustule in Man.** *Semana Médica.* 1926. Jan. 28. Vol. 33. No. 4 (1672). pp. 165–194. [19 refs.]

This is an interesting study of the results of various methods of treatment of anthrax infection in a large number of cases. Of a total of 414 patients, 82 were treated with injections of specific antiserum, 17 with normal bovine serum, 192 with peptone (5 per cent. Witte's peptone in distilled water with 0·5 per cent. NaCl, sterilized by heating for 15 minutes at 120° C., filtered and sealed in ampoules), starting with 10 cc. later 30 cc., twice or three times daily intragluteally injected, 88 with peptone and antiserum; 13 received symptomatic treatment only. The remaining 20 were subjected to various measures, such as antistreptococcus serum, neosalvarsan, etc. The subjoined table gives a summary of the results, the third column, denominated Revised Mortality, giving the percentages after excluding those patients who died within 48 hours of beginning treatment.

It will be seen that the best results were obtained by the peptone injections intramuscularly. When the intravenous route was attempted alarming symptoms were produced—headache, cough, palpitation, and if given otherwise than slowly there were fever, shivering, respiratory and circulatory distress with nausea or vomiting.

| Treatment. ... | No. of cases. | Mortality. | Revised Mortality. |
|---|---|---|---|
| | | per cent. | per cent. |
| Antiserum alone ... ... | 82 | 15·85 | 10·38 |
| Ditto+some other treatment ... | 141 | 19·14 | 12·97 |
| Normal Bovine serum ... ... | 17 | 29·41 | 25·00 |
| Ditto+other treatment ... | 33 | 21·21 | 18·75 |
| Peptone alone ... ... ... | 192 | 10·93 | 9·04 |
| Ditto+other treatment ... | 280 | 12·85 | 9·29 |

H. Harold Scott.

WEINBERG (M.). Enquête sur les traumatoses, et en particulier sur la gangrène gazeuse, chez les blessés du front marocain. [**Inquiry on Wound Infection, especially Gaseous Gangrene, in the Wounded on the Moroccan Front.**]—*Bull. Soc. Path. Exot.* 1926. Feb. 10. Vol. 19. No. 2. pp. 106–115. [1 ref.]

Infection of wounds with anaerobic organisms is found among the natives of Morocco. Dr. CRISTIANI at Fez has seen six cases of tetanus in country Arabs, and of gaseous gangrene has treated about three cases yearly. In fact gaseous gangrene is so familiar that a severe wound of a limb is bound firmly to prevent the entry of air, and the infection is designated by the Arabs by a word which means wind. Many cases of both tetanus and gaseous gangrene have occurred in wounded soldiers in the recent campaign; the organisms correspond fairly closely with those isolated in Europe in the War of 1914–18. Antigangrenous serum has proved very effective.

A. G. B.

BLOEDORN (W. A.) & HAKANSSON (E.). Some Observations regarding Culebra [Virgin Islands].—*U.S. Nav. Med. Bull.* 1926. July. Vol. 24. No. 3. pp. 534–544. With 8 figs. on 5 plates.

BUTLER (C. S.). The Medical Needs of the Republic of Haiti at the Present Time.—*U.S. Nav. Med. Bull.* 1926. Apr. Vol. 24. No. 2. pp. 269–279. With 2 figs. [8 refs.]

CAMPBELL (George). Focal Infections.—*Jl. Port-of-Spain Med. Soc. for 1924.* pp. 72–85.

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## REVIEWS AND NOTICES.

LANGERON (Maurice). Mycétomes — Oosporoses — Sporotrichoses — Blastomycoses.—[**Mycetomas — Oosporoses — Sporotrichoses — Blastomycoses.**]—*Roger—Widal—Teissier. Nouveau Traité de Médecine.* Fasc. 4. pp. 445–534. With 40 text figs. [Paris: Masson et Cie.]

Under the term Mycetomas are included inflammatory tumours caused by the proliferation of parasitic fungi in the tissue and characterized by the presence of grains formed of masses of mycelial filaments. While the foot is the most frequent situation of a mycetoma, any other part of the body may be attacked. Mycetomas are divided into two groups: Maduromycoses, in which the grains are formed of large septate hyphae, the causal fungus being a *Hyphomycete*; and Actinomycoses, in which the hyphae forming the grain are very fine and aseptate, the fungus being a species of *Microsiphonales*. Each of these groups is subdivided according to the colour of the grains into black, yellow, and red sections. Detailed consideration of each of these sections follows, and all fungi known to cause mycetomas are dealt with more or less fully. Paramycetomas, or tumours of fungus origin, but which do not form grains, the essential characters of true mycetomas, and Pseudomycetomas, which are tumours clinically simulating mycetomas but not of fungus origin, are also dealt with. Finally, there is a general discussion of the plurality of the causal fungi, the saprophytic origin, pathology, diagnosis, prognosis, treatment and prophylaxis of mycetomas.

The section on Oosporoses treats of those affections due to Microsiphonales, but which are not mycetomas, actinomyces or tuberculoses, in a manner similar to that on mycetomas. Classification is based on the proposals of FOULERTON, and CHALMERS and CHRISTOPHERSON. The *Microsiphonales* are divided into two genera, *Actinomyces* Harz 1877, and *Actinobacillus* Brumpt 1910. Actinomyces is divided into two sub-genera, *Euactinomyces* n. sub. gen., and *Cohnistreptothrix* Pinoy 1911; and *Euactinomyces* is again subdivided into *Parasitici* and *Saprophytici*, the former being split up into the sub-sections *majores*, *minores* and *breviores*. Under these divisions a list of all the known pathogenic species is given.

On the same general plan Sporotrichoses are dealt with. These are generalized mycotic infections due to fungi of the genera *Sporotrichum* Link 1809 and *Rhinocladium* Sacchardo and Marchal 1885.

Blastomycoses is a vague general term given to those infections caused by pathogenic fungi which reproduce by budding, although the budding, or yeast, stage may be only a phase in their life-history. Thus, a bewildering variety of different clinical conditions and of parasites of widely different systematic position comes within the range of this section. The parasites dealt with belong to the genera *Debaryomyces*, *Saccharomyces*, *Willia*, *Endomyces* (*Saccharomycetaceae*); *Cryptococcus* and *Monilia* Gmelin 1791 (*Hyphomycetes*, *Blastosporaceae*); and *Mycoderma* (*Hyphomycetes*, *Arthrosporaceae*). In this section the infections are arranged under a clinical classification according to localization, as the number of different causal fungi makes a satisfactory etiological classification impossible. The principal divisional headings are Blastomycoses of the mucosa, Cutaneous Blastomycoses, Blastomycoses of the bones, Visceral Blastomycoses, Generalized Blastomycoses, and Blastomycetic tumours. In spite of the immense number of species considered, it is concluded that not more than eight species are to be regarded as seriously pathogenic. As in the other sections, diagnosis, prognosis, treatment and prophylaxis are discussed in a general manner.

It is impossible to speak too highly of this admirable work. It contains all the essential facts on the subjects dealt with in a concise form devoid of masses of superfluous detail. The arrangement of the subject matter is such as to ensure the minimum of trouble in obtaining information on any particular parasite or affection. The difficult questions of nomenclature

and classification are handled in a masterly manner which gives little indication of the almost hopeless confusion met with in the literature on the subject. The mode of expression throughout, as in all Langeron's papers, is extremely clear and direct and renders the reading pleasant as well as instructive.

P. Tate.

LANGERON (Maurice). Intoxications par les champignons. [**Mushroom Poisoning.**]—*Roger–Widal–Teissier. Nouveau Traité de Médecine.* Fasc. 6. pp. 493–513. With 2 text figs. [Paris: Masson et Cie.]

In this short article of 21 pages M. Langeron gives from the point of view of the ordinary medical man a clear and valuable account of the mushrooms and toadstools and their allies that the practitioner in France —or anywhere in Europe—is most likely to encounter in cases of human poisoning.

Although poisoning by indiscriminate fungal meals is not rare in England, it would seem to be much more common and oftener to have fatal results in France. In 1912 there were 300 cases reported in France, with 100 deaths. By far the most deadly fungus—responsible for about 90 per cent. of the fatal cases—is *Amanita phalloides*. Apart from the more or less toxic effects that may be brought about by idiosyncrasy in the eater, indigestion, long storage or putrefaction, certain fungi contain specific poisons. On this fact ROCH has based his classification of poisonous fungi. M. Langeron has followed ROCH's classification, somewhat modified, and groups his fungi under the following heads :—

(1) Deadly. *Amanita phalloides*, *A. phalloides* var. *verna* and *A. virosa*. After an incubation period of 8–40 hours, these give rise to a choleraic condition, usually ending fatally with fatty degeneration and necrosis of the liver.

(2) Dangerous. *Amanita muscaria* and *A. pantherina*, giving rise, after an incubation of 1–2 hours, to either gastro-intestinal disturbance or delirium followed by stupor. Seldom if ever fatal.

(3) Irritant. A considerable number of species of different genera, giving rapid rise to gastro-intestinal disturbance, which soon subsides. Very rarely fatal.

(4) Haemolytic. *Morchella esculenta* and *Gyromitra esculenta*. If these are eaten quite fresh the helvellic acid contained may cause haemolysis, with jaundice and haemoglobinuria, accompanied with acute gastro-intestinal disturbance.

(5) Excitant of unstriped muscular fibres. *Claviceps purpurea*. Symptoms of ergotism.

(6) Fungi ordinarily edible, but become more or less poisonous in various ways by alteration.

The classification is neither strictly logical nor strictly scientific, but for the end in view practical enough. Sufficient details for the ordinary practitioner are given of the diagnoses of the more virulent species of fungi, and of the characteristic syndromes, pathological anatomy, pathogeny, prognosis and treatment of cases of poisoning. The medico-legal aspect of poisoning by fungi is briefly touched upon. Under prophylaxis, M. Langeron utters warnings against trusting to popular tests as to the innocence of fungi. He lays proper stress on teaching children to recognize the characteristics of the group to which the deadly Amanitas belong and emphasizes the desirability of every medical man knowing them. With this paper of M. Langeron at hand no French-reading medical man need remain ignorant of what may at any time prove most valuable information. This knowledge is just as desirable in England as in France, and fortunately, as far as the purely English reading practitioner is concerned, it is available, for Mr. J. RAMSBOTTOM's paper on "Edible and Poisonous Fungi" (*Procs. Roy. Soc. Med.*, XVIII (1925), pp. 13–26) covers practically the same ground.

A. T. Gage.

SEYFARTH (Carly). Die Malaria. [**Malaria.**]—*Handbuch d. Speziellen Pathologischen Anat. u. Histologie.* 1926. pp. 177–248. With 18 text figs.

In this article Dr. C. Seyfarth has given a concise and clear account of the morbid anatomy of malaria, based on his own experience and on that of pathologists of many nationalities.

The blood and the various organs are dealt with systematically, and the pathological changes, macroscopic and microscopic, are carefully described; the section on the morbid anatomy of the brain is particularly good. There is a short appendix on blackwater fever. The very full bibliography, including nearly all the recent publications on the subject, will be very useful.

The author remarks that, since the early work of VIRCHOW, MARCHIAFAVA, CELLI, BIGNAMI, and others, attention has been chiefly concentrated upon the aetiology and parasitology of malaria, and that too scanty interest has been taken in its morbid anatomy. This view seems rather too sweeping, and does not do justice to the careful investigation of the pathology of the disease that was made in many parts of the world before 1914. Certainly the war gave many pathologists the opportunity of studying malaria, with abundant *post mortem* material, and in Europe, especially, there was the further advantage of access to numerous cases of recently acquired and uncomplicated malaria. In the tropics, a large proportion of the malarial cases that are submitted to *post mortem* examination are complicated by the presence of other diseases; but in spite of this difficulty, the essential features of the pathology of malaria were well worked out by many competent tropical pathologists before the war aroused a more widespread interest in the subject.

H. J. Walton.